I.

GEORGE MOREWOOD LEFFERTS.
1846—1920

BY D. BRYSON DELAVAN, M. D.,

NEW YORK.

The passing of George Morewood Lefferts marks the close of an important era in the history of laryngology, as well as the departure of one whose lifework exercised no little influence in the development and dissemination of the knowledge of his art.

Student, pioneer, organizer, instructor, practitioner and always leader, we may with profit to ourselves study the incidents of his brilliant and eminently useful career, and analyze the elements of character and of disposition which contributed to its success.

Dr. Lefferts was born in Brooklyn, N. Y., February 24, 1846. His grandfather was of a distinguished Dutch family of New Amsterdam, the original member of which came over from Holland in 1660. The maternal grandfather was Gilbert Allen, an oldtime merchant of New York, but, like the grandmother, of the purest and best of the original New England stock, one branch dating back to the Mayflower.

Marshall Lefferts, his father, was by profession an electrical engineer in New York, and the inventor of improvements in telegraphy. He was a man of strong character and remarkable physical perfection, possessed of characteristics which easily placed him in successful command as Colonel of the 7th Regiment N. G. S. N. Y., then as now the best regiment of militia in the United States. At the time of the Civil War, with the Capital practically unprotected, the 7th Regiment was the first to reach Washington, thus relieving the keen anxiety of President Lincoln, who for eleven trying days and nights had watched from the windows of the White House the campfires of the enemy just across the Potomac. As a child the writer was one of the deeply moved throng which cheered the gray-clad regiment as it made its splendid march down Broadway, with Col. Lefferts at the head. Twice in these later days, under other fine leadership, has he witnessed this same stirring pageant, but neither these nor others of their kind have dulled for him the thrill of that early memory. So well had the men of the 7th been instructed and trained for military duty under the leadership of Col. Lefferts, that of the 1100 composing its roster 620 were quickly selected as officers and placed in command of less experienced troops. a service nobly repeated in the Spanish and World Wars.

The history of the father was exemplified in that of the son, who, no less a captain of men, trained an army of leaders in the march of scientific progress. The inventive genius, the initiative, the ability and the power to command which were so conspicuous in the son were clearly inherited.

Dr. Lefferts' preliminary education was received in the public schools of New York, and he was graduated from the College of the City of New York in 1867. He at once entered the College of Physicians and Surgeons, Columbia University, receiving the degree of M. D. in 1870. In 1869 he received the honorary degree of A. M. from Dickinson College, Carlisle, Pennsylvania, and in 1901 the degree of M. S. from Columbia University. Upon graduating in medicine he filled a short internship in Bellevue Hospital, New York, passing from that to an eighteen months' service at St. Luke's Hospital.

Following the fashion of the day, he then went to Europe, where he spent two years of well directed, active work at the

George M. Lefferts
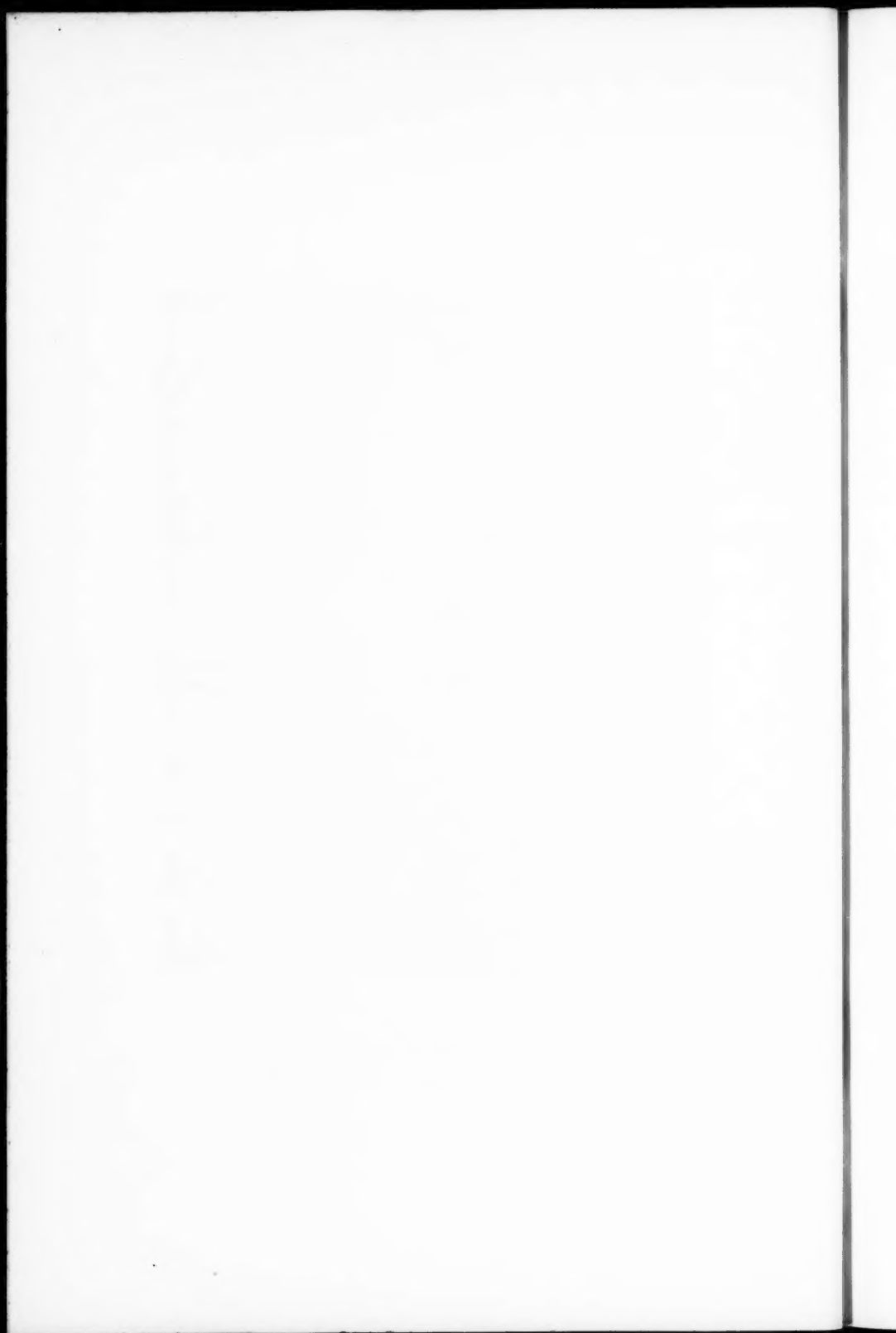
laryngologic clinics of London, Paris and Vienna. His earnestness and ability soon recommended him to his instructors, who recognized in him a pupil sure to do them credit and were accordingly interested in him. This was particularly true in Vienna, where he attended the clinic of Professor von Schrötter, and later that of Professor Karl Stoerk, of the Imperial University. Upon the latter he made such a favorable impression that he was soon appointed Chief of Clinic, which highly creditable and advantageous position he held during the years 1871-73, until his departure from Vienna, being the first young American to have attained that coveted distinction. Upon completing his studies at Vienna he received from Prof. Stoerk a testimonial of which the following is a translation:

"The undersigned hereby certifies that Dr. G. M. Lefferts has frequented his Laryngoscopic Clinic since October, 1871, with such zeal and success and has shown during his time of study such skill that he gave him the position of his assistant when it became vacant.

"During his time of service Dr. Lefferts has repeatedly performed both operations for laryngeal polypi and other difficult intralaryngeal operations with such skill that the undersigned can certainly term him one of the best laryngoscopists. Dr. Lefferts has won this acknowledgment particularly from the fact that he represented me so efficiently in my courses on laryngeal diseases and laryngoscopy, that during my absence he conducted both the instruction of my students and the clinical treatment of the patients quite independently and to my extreme satisfaction up to the present time.

"The only thanks for his efficient services that I can possibly offer him is to acknowledge them in documentary form and to recommend him most warmly to every one on account of his great attainments in medical science.

<div style="text-align:center">

(Signed)   "DR. KARL STOERK,

"Professor of Laryngoscopy and Laryngeal Diseases,

Imperial University of Vienna, etc.

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"VIENNA, March 13, 1873."

Both of his Viennese instructors regarded his subsequent success with genuine pride. They were his warm friends for life. The same was true of those in London and Paris.

His student companions of those early days have always reverted with pleasure to the delightful impression everywhere made by him.

In 1873 he returned to New York, where he at once entered upon the practice of his profession, specializing upon diseases of the upper air passages, and finding quite enough in that department to fully occupy his time and satisfy his ambition.

The same year he was appointed extramural teacher of laryngology at the College of Physicians and Surgeons, and in 1875 full instructor; later in the same year, clinical lecturer, and in 1876 clinical professor of laryngology and rhinology. This position he held until 1904, a period of service of over thirty years. Upon his retirement he was made Professor Emeritus by the trustees of Columbia University.

Dr. Lefferts regarded his professorship as the highest honor and the greatest opportunity of his life. Notwithstanding his other professional interests and accomplishments, this one was always his chief interest and pride. To it he devoted his best energies, unsparing of time, labor or financial outlay. Year after year his lectures were revised with scrupulous care, new illustrative material of every possible sort added, and improved arrangements made for the securing and demonstration of the best clinical material. The faithfulness of his attendance is attested by the fact that he missed but one lecture appointment in a period of more than twenty years.

The lectures were conducted with true military promptness and precision. Every detail was prepared and arranged in advance with the most minute care. No stage setting could have been more elaborately planned. Promptly upon the appointed hour and at the stroke of the bell, followed by his staff, he appeared before the class, faultlessly attired as for a court reception, his countenance radiating the earnestness and the enthusiasm which possessed his mind. He was the personification of the ideal instructor, impressing his audience with the thought that he held matters of great value and importance, which he was earnestly anxious to share with them and which he strove with all his power to impart.

As a lecturer he was clear, emphatic, persuasive, and, if sometimes a little sententious, always so with the object of being impressive, never failing to hold the interest of his hear-

ers and to send them away having understood and mastered
what he intended them to know.

His assistants were carefully selected. Each was trained to
the performance of a special duty, which duty, at the proper
time, was expected to be executed with the promptness and
accuracy characteristic of the professor himself.

For the illustration of the lecture everything was added in
the way of object-teaching that his remarkable ingenuity could
devise.* Each particular topic was illustrated by a series of
charts, splendid pictures in water color, showing every type
and phase of the subject at hand. These were of large size,
made by a specially trained and skillful artist under Dr. Lef-
ferts' direct personal observation, from life and from models
taken from the best authorities, the subject of each being
clearly inscribed upon it. During the lecture the charts per-
taining to the subject were displayed in full view of the class.
To the collection of charts, which included upwards of 500
specimens, there was added a full collection of papier-maché
models of the larynx, illustrating the anatomy and the various
phases of the pathology, together with numerous well mounted
pathologic specimens. The special instruments and forms of
apparatus pertaining to the subject were displayed and as far
as necessary described. Of these, as with the charts, there
was a comprehensive and lavish outfit, carefully selected, in-
structive, and collected regardless of expense.

But this was not all. Dr. Lefferts was a remarkable draughts-
man. His blackboard drawings, with colored chalk, were su-
perior to any of his time except those of the late Professor
John C. Dalton. Wherever desirable the larynx or pharynx of
the patient to be demonstrated was illustrated on the board in
the presence of the class, the process of the drawing going on
with the description of the case.

These so-called "lectures" were by no means what is under-
stood as "didactic." The lecture itself, fine as it was, was but
a necessary preliminary, explanatory of the principal feature
of the exercise. This was the actual exhibition of patients.
From the resources of himself and his many assistants, each
of whom conducted large outside clinics, numerous cases illus-

*See "Appendix," page 381.

trative of the subject in hand, suitable for exhibition and easy
of demonstration, were sent to the college, the best selected,
and these demonstrated personally by the Professor, a line of
students being formed behind him in the amphitheater, each
student in turn standing in a position close to the side of the
demonstrator from which a clear view of the image in the
mirror could be obtained. Time enough was allowed to enable
each one to recognize the lesion shown, a general idea of
which had already been given by the lecture and by the draw-
ings, models and charts.

At the beginning of the annual session a printed scheme of
the lectures was handed to the members of the class. Upon
a printed folder was given the number, date and title of each
lecture, with a comprehensive synopsis of its contents. By this
means the student was apprised of the nature and scope of
the course and was enabled in advance to prepare himself for
each lecture by such reading and clinical observation as time
and opportunity allowed. Subsequently the synopsis furnished
an excellent basis for review and for the refreshment of the
memory. In addition to this, pamphlets were distributed in
the course of the lectures, some of which explained the differ-
ential diagnosis between the principal laryngeal diseases, while
others gave full and explicit directions for their treatment.

Supplementary to the regular lectures was a course of special
instruction in the practical demonstration of the upper air
passages. This was carried on under the guidance of the Chief
of Clinic and a corps of specially trained assistants. The class
was divided into groups of six, and six lessons were given to
each group—three lessons a week for a fortnight, the time
devoted to each lesson being one hour. A special room was
equipped for the purpose of this instruction, in which stalls for
each student were provided, and these were furnished with
the illuminators, models of the human head and the other
appurtenances necessary for the examination of patients.*

The course consisted of instruction in the use of the instru-
ments necessary in demonstrating the upper air passages,
including pharyngoscopy, laryngoscopy, anterior and posterior
rhinoscopy, and the examination of infants. Life size dissected

*See "Appendix."

models of the human head were supplied. These were invaria-
bly used preliminary to exercises upon the living subject, for
the purpose of instructing students in the technic of the man-
agement of the head mirror and in the rules for the application
of laryngoscopy, pharyngoscopy and posterior rhinoscopy.
This method—employed by Türck and by the best of the other
old masters—enables the student to first acquire the principles
of the technic of the examination without his attention being
disturbed by the discomforts of the living subject. The prin-
ciples having been mastered, normal subjects were at once sup-
plied, and, later, more extended courses were given in the
examination and diagnosis of pathologic conditions.

Scrupulous care was taken to superintend the work of each
individual student and to see that the rules taught were thor-
oughly explained, understood and mastered. At the close of
the course of instruction the men were well grounded in the
rudiments of the work and reasonably prepared for its further
advancement.

The admirable working of this system of practical instruc-
tion began to be impaired when the number of students in a
group was increased to eight; when the number became twelve
it was almost impracticable; and when, at last, the order went
forth that the class should number twenty, the time for the
method had gone by. Students who had profited by the course
of lectures and practical instruction herein described graduated
with a profound respect for laryngology and a clear knowledge
of its principles.

The system of instruction thus instituted, organized and
conducted has been described in detail because never in the
department of laryngology had there been a similar course.
Others had taught the specialty, some wonderfully well; but
nowhere, either in this country or in Europe, had the under-
graduate ever had the subject placed before him with such
clearness, such comprehensiveness and such practical success.

The department thus founded at the College of Physicians
and Surgeons was the result of lavish expenditure and of
patient, unremitting labor covering the period of a generation
by a man peculiarly endowed. He left it a perfect thing, a
splendid model for the whole system of clinical instruction in

general, and as such alone it was most desirable that it should have been permanently preserved.

In 1904, completing an active service of over thirty years, Dr. Lefferts resigned his professorship and was at once elected Emeritus Professor by the trustees of Columbia University and at the following commencement was awarded the honorary degree of "Master of Science" (M. S.). The minute entered upon the archives of the institution reads as follows:

"The Faculty of the College desires to express its high appreciation of the distinguished services of Dr. George M. Lefferts, which through three decades he has rendered to the institution. The organization and maintenance of an important, practical, clinical department in the College—eagerly sought by the students of successive generations and a model of efficient administration—we recognize as a large achievement in the career of a practitioner. This long and faithful service and the generous gift to the College of a valuable collection of illustrative specimens and charts are held in high appreciation by his colleagues. This collection, by resolution of the Trustees of Columbia University, adopted November 2, 1903, is known as the 'Lefferts Museum of Teaching Apparatus in Laryngology and Rhinology.' "*

Dr. Samuel W. Thurber, for many years Dr. Lefferts' associate, has prepared an elaborate "order-book" of the Lefferts lectures. This book is now deposited in the department of "Columbiana" in the Library of Columbia University. It presents in detail, drawn to scale, the precise position in the amphitheater of all the apparatus used in the illustration of each lecture, together with photographs showing the amphitheater so prepared and equipped. A full collection of these pictures is given in the appendix to this memorial. It is an interesting and instructive exhibition in itself and, incidentally, an example of the finished exactness characteristic of the man.

When Dr. Lefferts returned to New York, in 1873, there was no society of laryngologists in any city in the world. In October of that year the New York Laryngological Society, now in successful existence for half a century, was founded by Dr. Clinton Wagner. Dr. Lefferts was one of its

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*See "Appendix."

charter members. This society, in 1885, became the Section in Laryngology of the New York Academy of Medicine.

In 1878, Dr. Lefferts was one of the founders of the American Laryngological Association. His address delivered at the opening of the first meeting clearly and eloquently set forth his ideals of the aims and functions of such a society. It was he who originated the name of the Association, and, as its first secretary, was chiefly instrumental in its successful organization and in establishing for it those high ethical and scientific standards under which it has flourished preeminent for more than forty years.

The development of his system of undergraduate instruction, and the part which he played as a pioneer in the establishing of not only the first, but by far the best, of the societies devoted to his specialty were by no means his only notable achievements.

His career in the field of clinical medicine was conspicuous. Prompt, energetic, efficient, a specialist skilled in every department of his art, his personality combined with the interest which he invariably manifested towards his patients caused his private practice and his public clinics to grow rapidly in numbers and in fame. He was appointed laryngologist to the then popular Demilt Dispensary, in 1873, in company with Dr. Charles McBurney. This department soon gained a name for itself, Dr. Lefferts serving it for six years.

It was at the Demilt Dispensary, in 1873, that Dr. Lefferts, with others, established a regularly organized system of instruction for graduates in medicine, nearly ten years before the founding of the New York Polyclinic and the New York Post-Graduate Schools.

He was the leading member and largely the organizer of the throat department of the New York Eye and Ear Infirmary, receiving his appointment in 1874 and holding it until 1891. He was appointed consultant to the outdoor department of Bellevue Hospital in 1886, and was consulting surgeon to St. Luke's Hospital from 1877 until 1901. These appointments, together with his large clinic at the College of Physicians and Surgeons, were filled by him with the utmost zeal. The amount of material at his disposal for his own observation and for use in the course of his lectures from his own clinics and those of

his numerous assistants was enormous. His extensive private and consultation practice embraced a clientèle of the very first order of influence and importance.

Always with the advance guard, Dr. Lefferts distinguished himself in the literature of laryngology. His original theses, in a style well written, terse and to the point, covered a variety of subjects and were valuable additions to the knowledge of the special topics with which he dealt.

In the editorial department of laryngology he was again a leader. In 1880 there was no journal in this country which represented our special department of medicine. Dr. Lefferts, in association with Dr. Louis Elsberg, of New York, Dr. J. Solis-Cohen, of Philadelphia, and Dr. Frederick Irving Knight, of Boston, founded a quarterly magazine which was named The Archives of Laryngology. This periodical furnished another exemplification of the controlling principle of Dr. Lefferts' life. What his ambition demanded and what he ever strove to attain was the best. In company with his distinguished collaborators, The Archives of Laryngology was made by far the best journal of laryngology that had ever appeared, in beauty of form and excellence of material surpassing anything abroad. Ably and generously conducted by its patriotic editors, at the end of four useful years it was discontinued by reason of lack of support, a grievous loss to the scientific world.

Dr. Lefferts was collaborator for the Archives de Laryngologie of Paris, and from 1884 to 1910 an active collaborator of the Internationales Centralblatt für Laryngologie, Rhinologie, etc., of Berlin. He was associated with a number of other foreign periodicals. His name also appears as contributor to various systems of medicine and surgery.

With that ever dominant desire to disseminate knowledge, one of his most useful missions was accomplished through the medium of abstracts, that is, the placing before the medical public the best of the new thought and ideas of the leaders of the laryngologic world. In the classic work of Sir Morell Mackenzie, published in 1880, there was given an invaluable bibliography, dating from the earliest times. In May, 1875, Dr. Lefferts, in the New York Medical Journal, initiated the publication of a series of laryngologic references accompanied

by abstracts of such articles as were worth while. In this particular department there does not appear to have been any one by whom he was antedated. In 1880 the abstract department was transferred from the New York Medical Journal to The Archives of Laryngology. By the time the Archives went out of existence the Index Medicus, which had been instituted by Dr. John S. Billings, took over the bibliography, while the abstracts were continued in the Centralblatt. Thus, with the books of Sir Morell Mackenzie, the contribution of Elsberg,* the current records of Lefferts, the Index Medicus and the continuation of Lefferts' work in the Centralblatt, THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, the Laryngoscope and other periodicals, the bibliography of laryngology is remarkably complete to date. Here, again, Dr. Lefferts was the pioneer. As collaborator with him for many years in this work, the writer well knows the importance which he attached to it and the tireless energy with which he carried it out.

Having thus sketched the life-work of Dr. Lefferts, let us now consider the personality of the man and the elements of character and of intellect which determined his success.

Early in life he had suffered a condition of lameness never entirely recovered from, and necessarily, to one of his sensitive nature and active temperament, a serious affliction. The eldest of seven children, the favorite of an indulgent family circle, one less resolute and of weaker moral courage might easily have failed of great accomplishment. Refusing to yield to the allurements of a life of indolent self-gratification, the burden of his disability was borne by him with the calmness of a philosopher and the fortitude of a brave man. Far from bringing bitterness and discouragement, it but served to stimulate his ambition and to quicken the energy of his intellect, as proved by the ever recurring testimony of his history; for, with every temptation to the contrary, neither excellent social position, ample means for gratifying his luxurious tastes, nor his well developed capacity for the enjoyment of the higher pleasures of life could divert him from his chosen calling or quench the enthusiasm with which he followed it.

*Trans. Am. Lar. Assn. vol i. 1878. "A Bibliography of American Laryngology."

Of unusually attractive personality, Dr. Lefferts was pos-
sessed of a countenance strikingly alert, intelligent and hand-
some—blonde, with features well modeled and refined but
nevertheless strong. The gentleness and cordiality of his
impulses, the readiness of his sympathy, the charm of his man-
ner, were such as to induce enduring friendships. The recol-
lection of his mirthful pleasantries and the inimitable radiance
of the smile which acclaimed them will long be treasured
among the happy memories of his friends. Bright and genial
in conversation, he was a welcomed companion and a charm-
ing host.

Inheriting elegant and refined tastes, he strove for perfec-
tion in all that he undertook, from faultlessness of attire and
of the surroundings of his daily life to every detail of his work.
One of the chiefest of his characteristics was the love of order.
Everything connected with his affairs, whether professional,
business or social, was carried out with the utmost regard for
system. Even the records of his routine work were models
of thoroughness and elegance, set forth with consummate clear-
ness and perfection of arrangement. With keen prevision,
nothing was left to chance. His watchword was "prepared-
ness." His whole life-work was an eloquent exemplification of
that idea. Of sterling integrity, he was in all things the soul
of honor.

Strong and positive by nature, his decisions were quickly
made and as promptly executed. His handwriting was unique
in its clearness and marked individuality. He was an excellent
draughtsman both with crayon and pen, and with the latter
often evinced a keen sense of humor. Having distinctly artistic
tastes, his judgment of art in its various departments was
excellent. Fond of travel, he spent many summers abroad, one
in particular in Japan at a time when fine specimens of its
antique art were still obtainable. It was then that he secured
the superb collection of ancient Japanese armor which, depos-
ited as his gift, is now one of the chief treasures of the Metro-
politan Museum of Art. The dinners given by him annually
to the members of his clinical staff at the old Delmonico's,
now things of the past, were to us one of the important events
of the year. He was a good horseman and greatly enjoyed
that form of exercise. During the season there were few days

which did not find him in the saddle either upon the bridle
paths of the park or in the ring of the riding club.

That he succeeded brilliantly in the work of his profession
was due to no mere chance of fortune or of meretricious favor.
The grace and talent that were his by fine inheritance he fully
recognized, and, recognizing, used and cultivated with untir-
ing industry and zeal, devoting them with unwavering fixed-
ness of purpose to the special object of his life. This, his pro-
fession, he mastered in all its details and by every possible
means. Clinical experience, wide reading, study, writing,
teaching, travel, intimate association with the most distin-
guished authorities at home and abroad: these were the weap-
ons with which he steadily fought his way to preeminence.
Excellent as a writer, of remarkable initiative as an organizer,
great as a practitioner, he himself would have wished us to
believe, as in fact we do, that his highest usefulness was at-
tained in the teaching of his beloved art. Indeed, the keynote
of his philosophy was expressed in his chosen motto: "Docendo
discimus"—by teaching we learn.

A gentleman by birth and breeding, his character, tempera-
ment and ability were such that in any department of effort
he would have been a leader, a highly efficient man of affairs.
If he could have left to the world but one legacy, the example
of the lifelong courage, industry and enthusiasm with which,
by all the varied means we have enumerated, he strove to
relieve the sufferings of humanity, would be to us a priceless
example, a rich inheritance.

Dr. Lefferts was married June 11, 1891, to Miss Annie
Cuyler Van Vechten, of Albany, New York, the daughter of
Abraham Van Vechten, Esq., an eminent lawyer of that place.

Miss Van Vechten had attained distinction in the social life
of her home and in Washington, where, during the presidency
of Mr. Cleveland, she was a frequent guest of his sister, Miss
Rose Elizabeth Cleveland, at the White House, and a brilliant
addition to its chosen circle. Following her marriage, Mrs.
Lefferts' residence in New York City included a period of
nineteen years. At the end of this time Dr. Lefferts' health
began to show signs of impairment. For thirty-seven years, as
student and practitioner, he had pursued continuously an
arduous professional career. Never physically robust, and

recognizing that the limits of his endurance seemed to have been reached, he wisely acquiesced, and withdrawing from all medical interests removed to his country seat at Katonah, in the Bedford Hills, New York. There, with Mrs. Lefferts, amid surroundings which he loved, he rested for ten peaceful years, cheered and sustained until the end by the companionship which had been the crowning happiness of his life.

Long a sufferer from cardiac angina, he died at his home September 21, 1920.

Dr. Lefferts was Fellow of the Academy of Medicine, New York; past president (1876), New York Laryngological Society; past president (1882), of the American Laryngological Association; past president (1891), of the Alumni Association of the College of Physicians and Surgeons; Honorary Fellow of the Laryngological Societies of London, of Vienna and of Berlin; and a member also of the Surgical Society of New York, American Medical Association, New York State and County Medical Societies, and the University and Riding Clubs of New York.
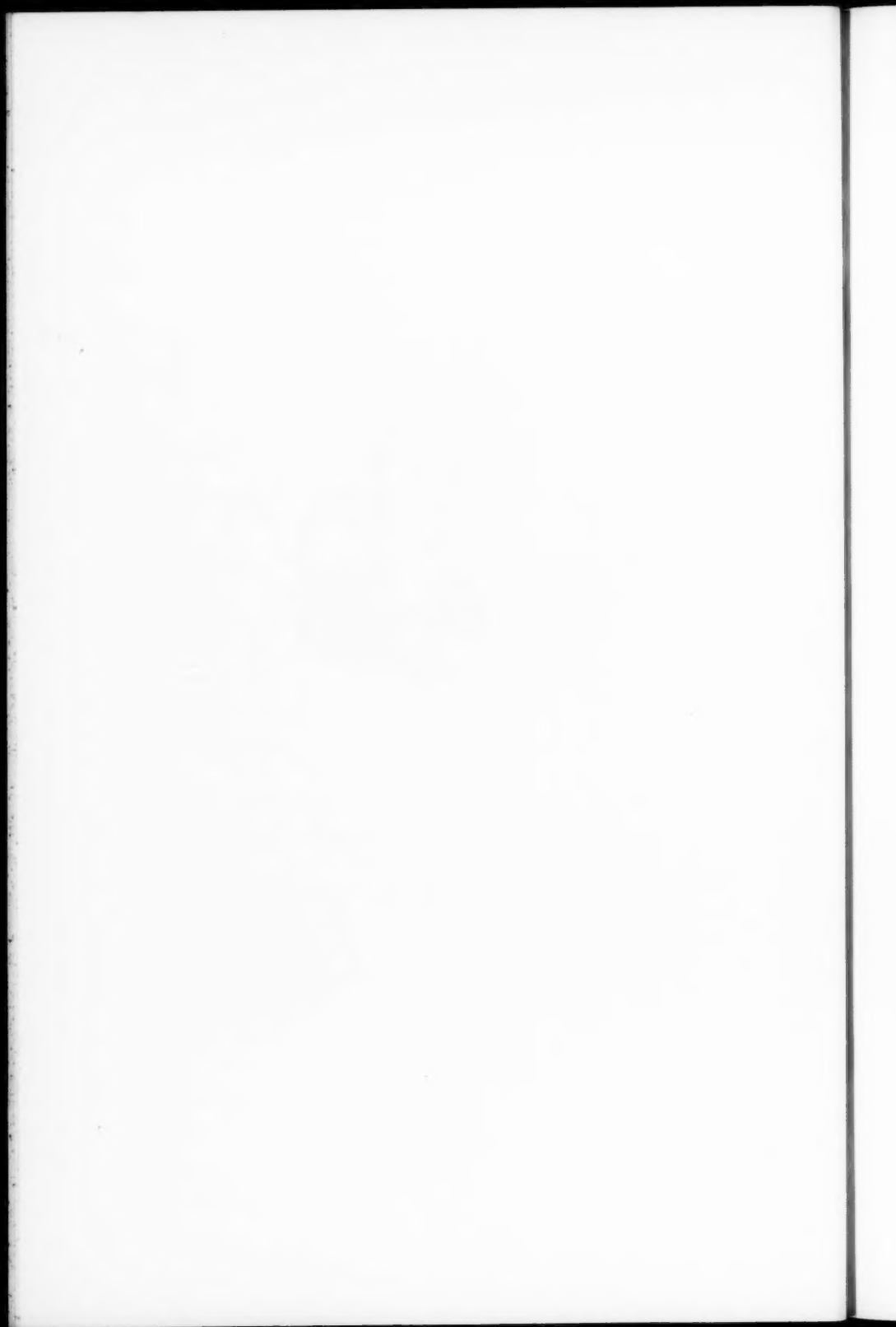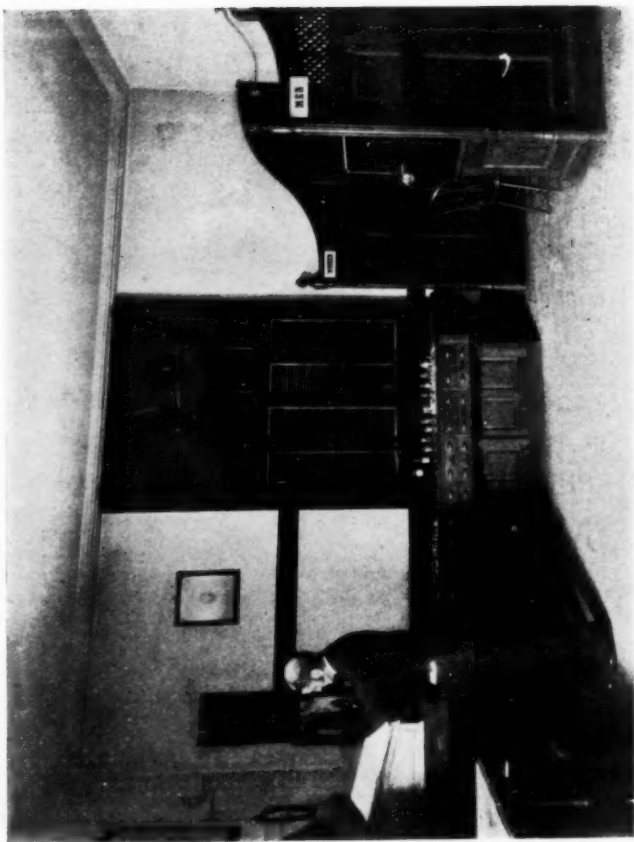
APPENDIX

THE LEFFERTS CLINIC

A COLLECTION OF HALFTONE ILLUSTRATIONS REPRESENTING
THE TEACHING EQUIPMENT OF

GEORGE MOREWOOD LEFFERTS,

A. M., M. D., M. S.

PROFESSOR OF LARYNGOLOGY AND RHINOLOGY, COLLEGE
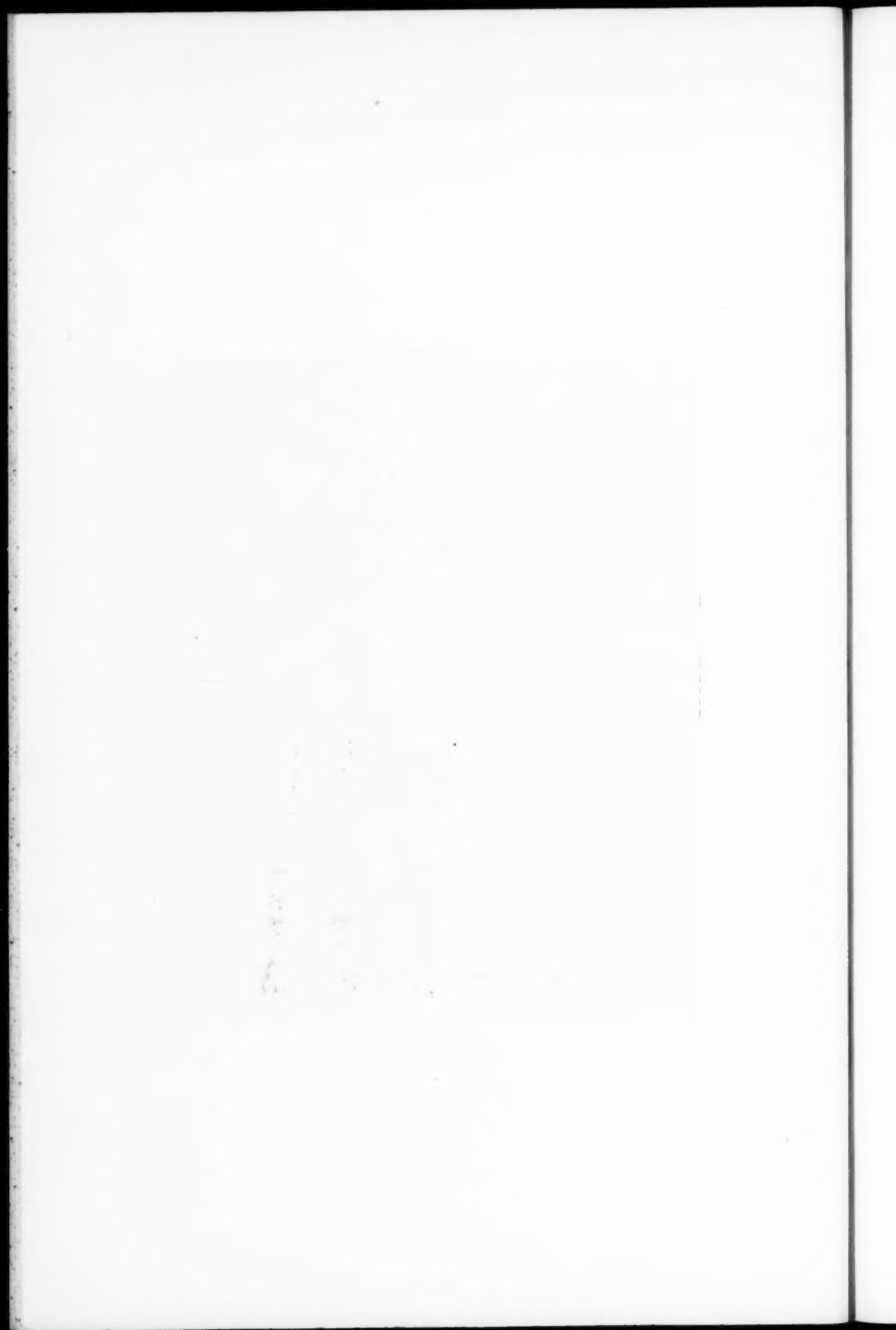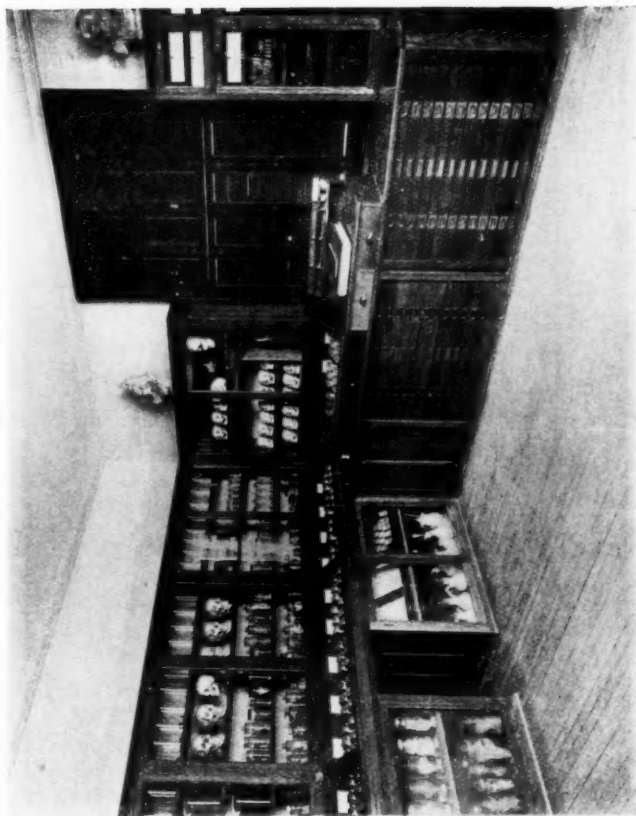OF PHYSICIANS AND SURGEONS, NEW YORK.

PROF. LEFFERTS.
SURGEONS' ROOM, VANDERBILT CLINIC.

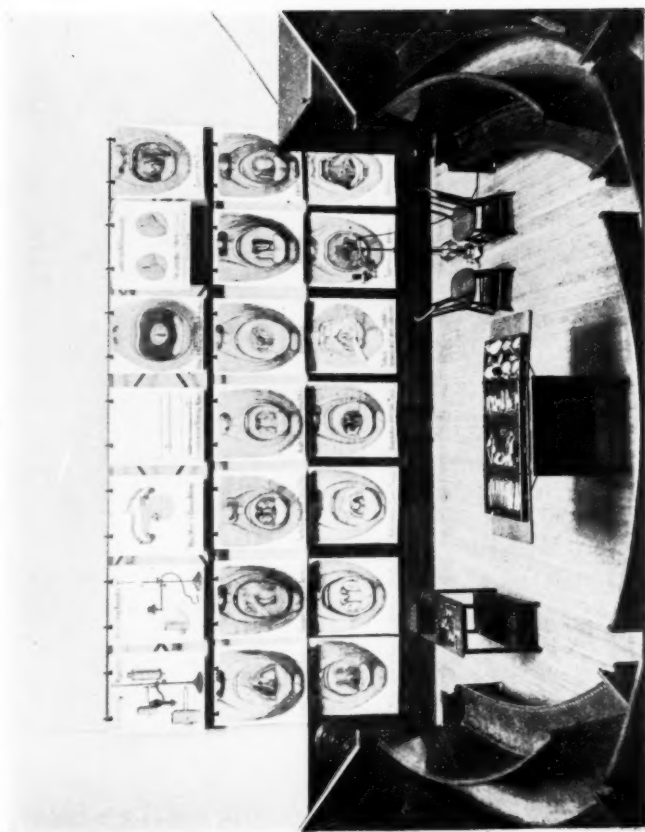CABINETS FOR EXAMINATION AND TREATMENT OF PATIENTS,
VANDERBILT CLINIC.

SECTION OF THE LEFFERTS MUSEUM OF TEACHING APPARATUS
IN LARYNGOLOGY AND RHINOLOGY.
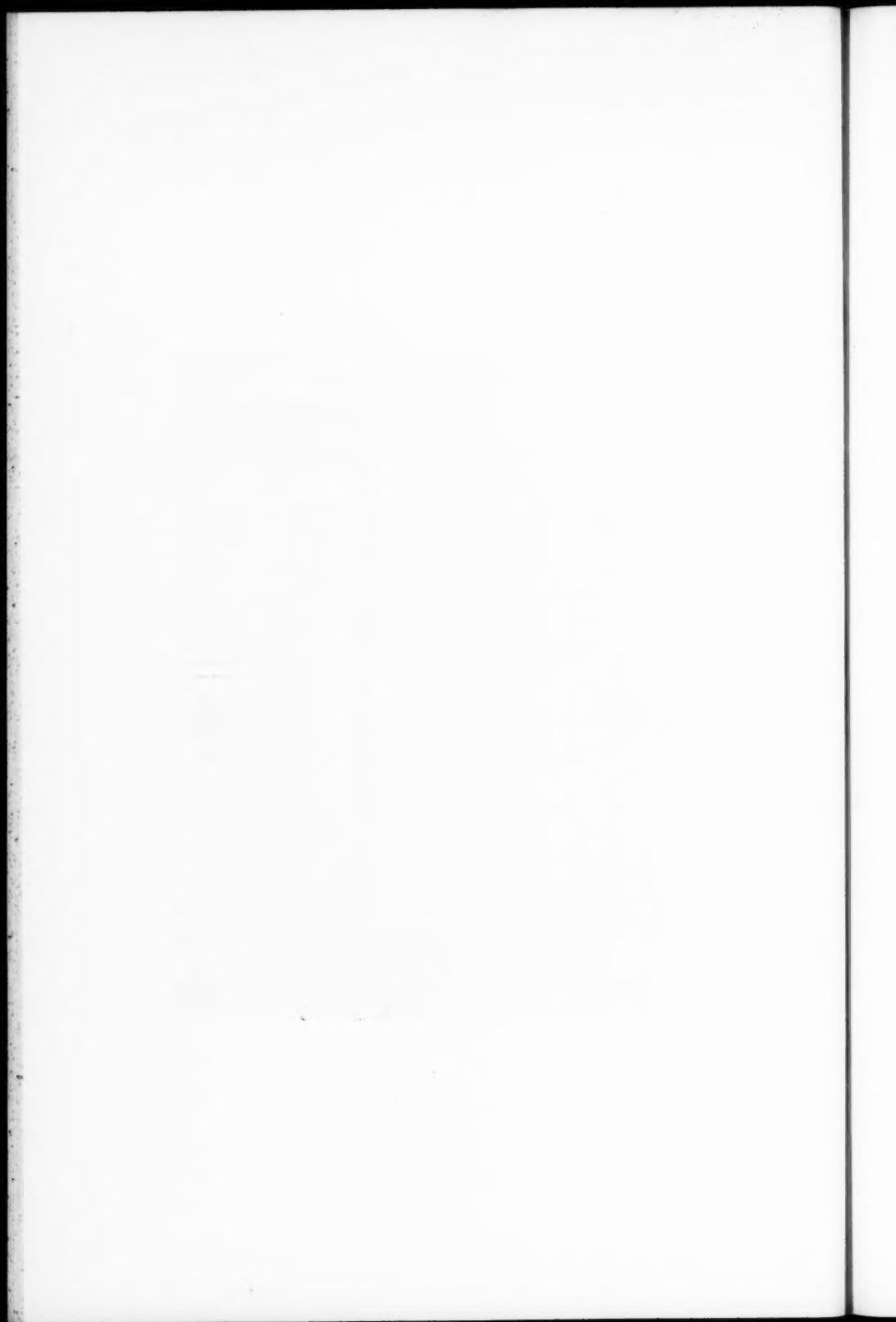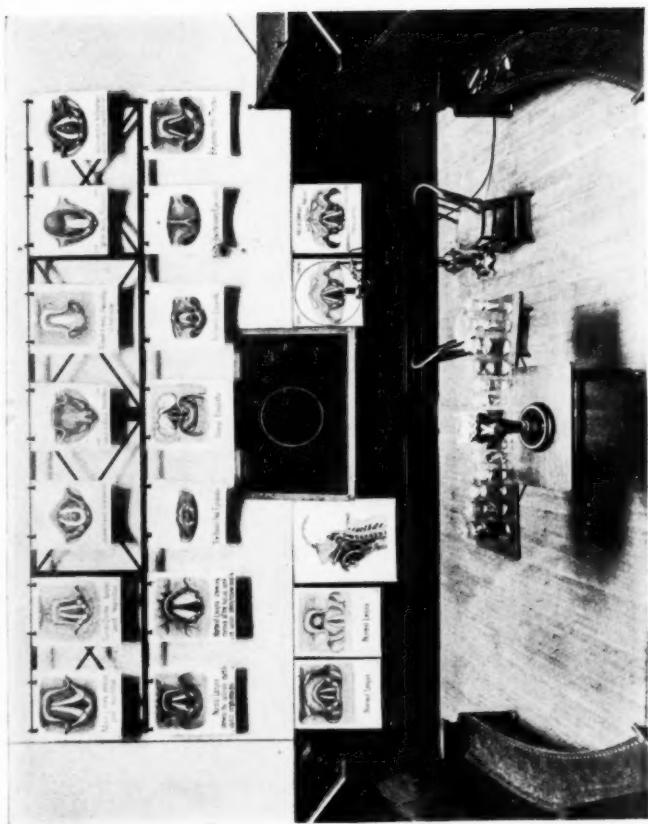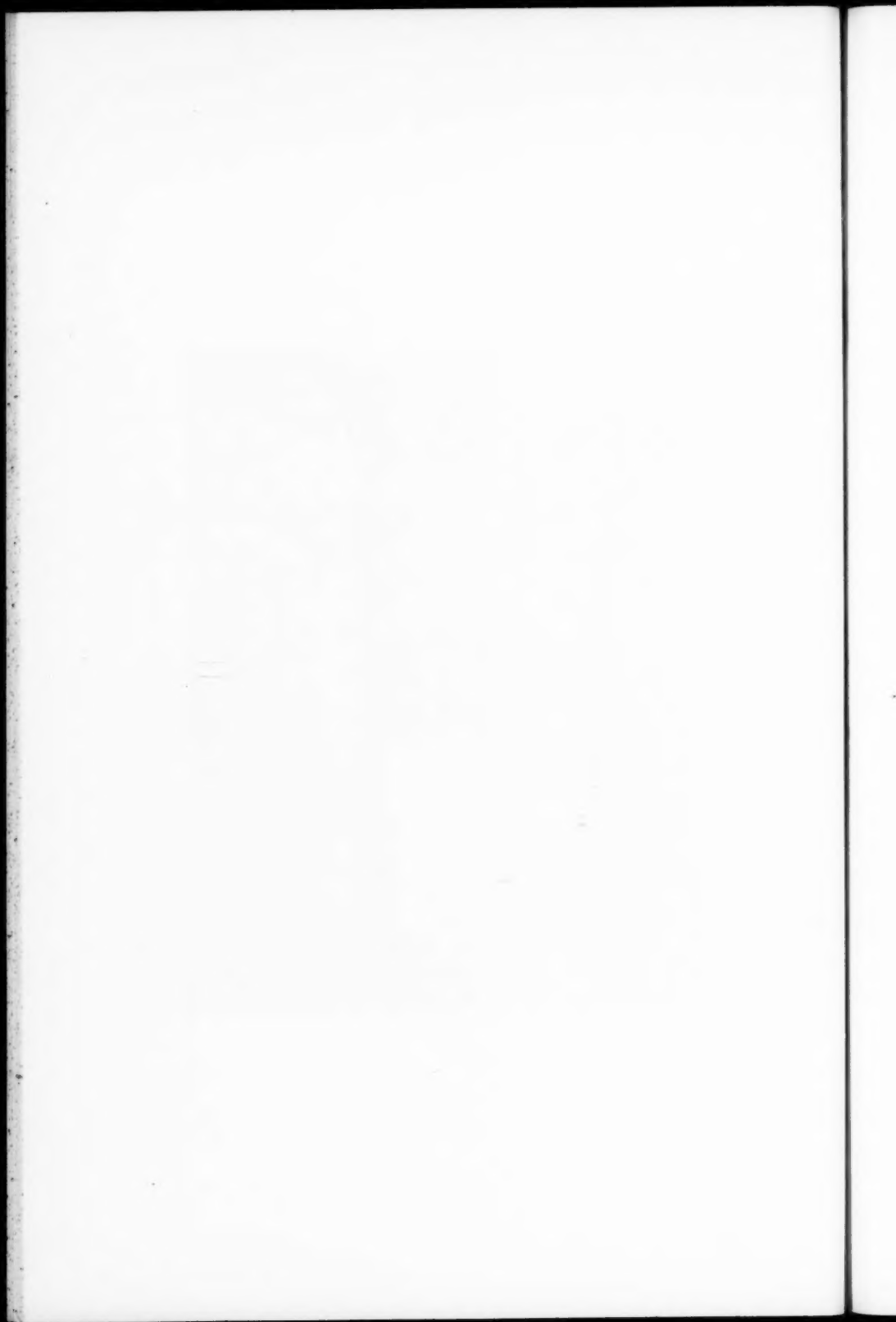
HALL OF PRACTICAL INSTRUCTION.

Lecture No. I.—EXAMINATION OF PHARYNX AND LARYNX.
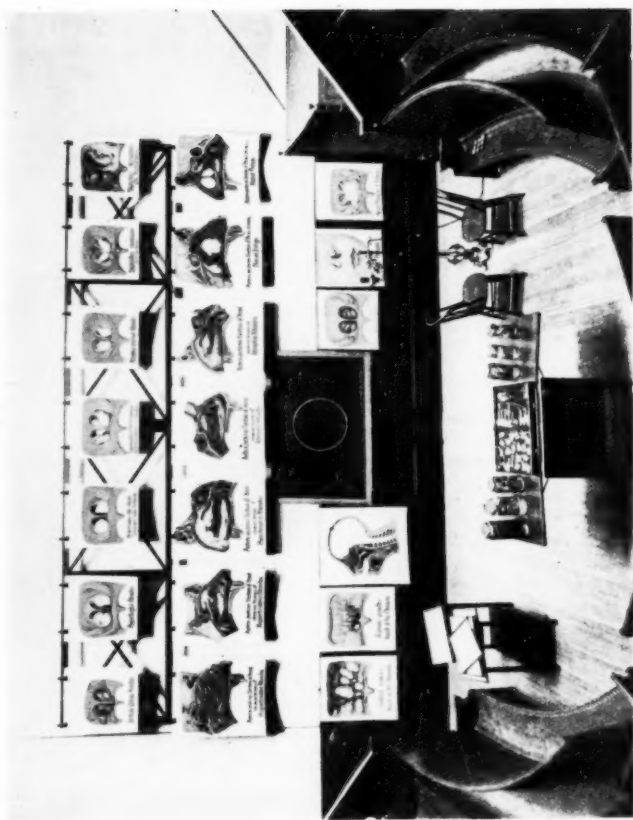
Lecture No. II.—ANATOMY OF THE LARYNX. LARYNGOSCOPY.

Lecture No. III.—ANATOMY OF NASAL PASSAGES. RHINOSCOPY.
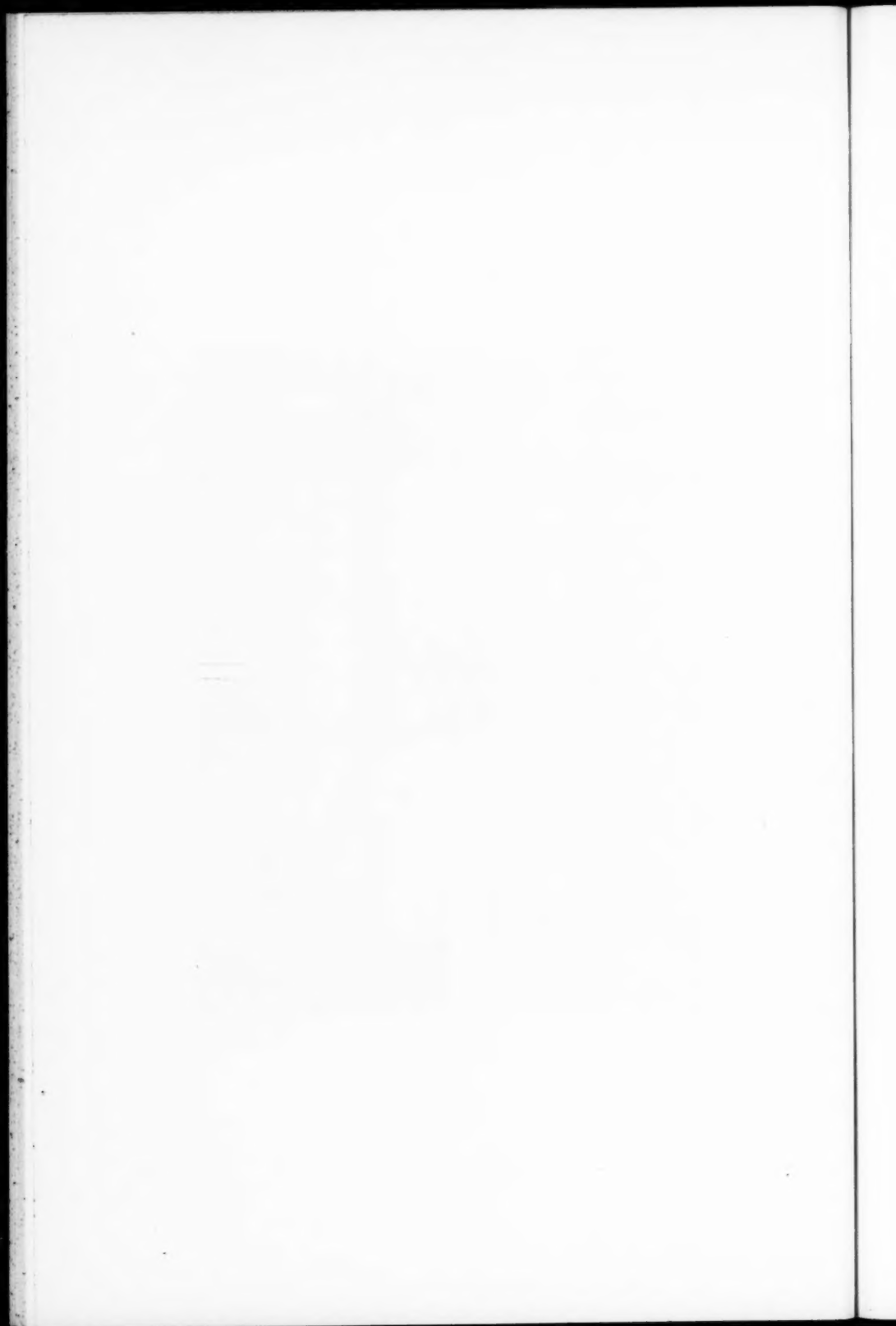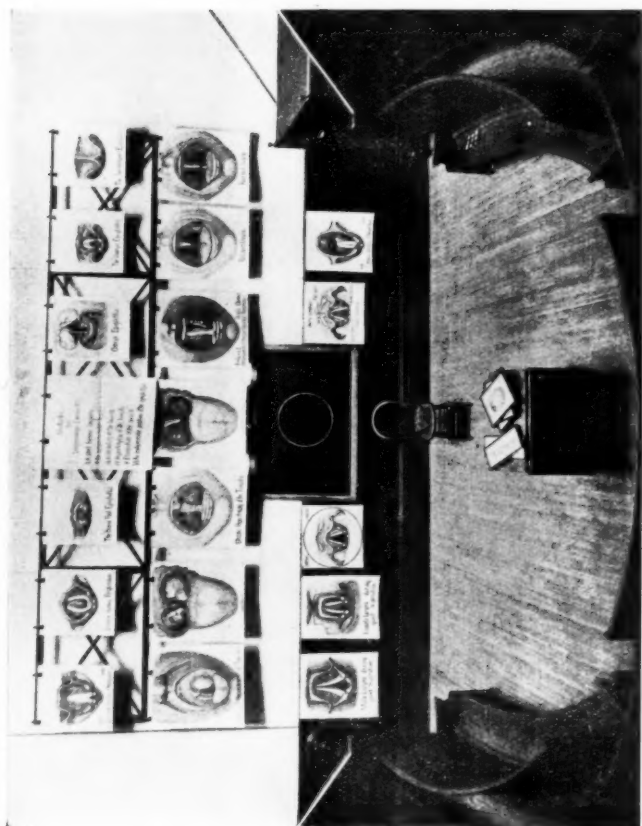
Lecture No. IIIA.—OBSTACLES TO A LARYNGOSCOPIC EXAMINATION.
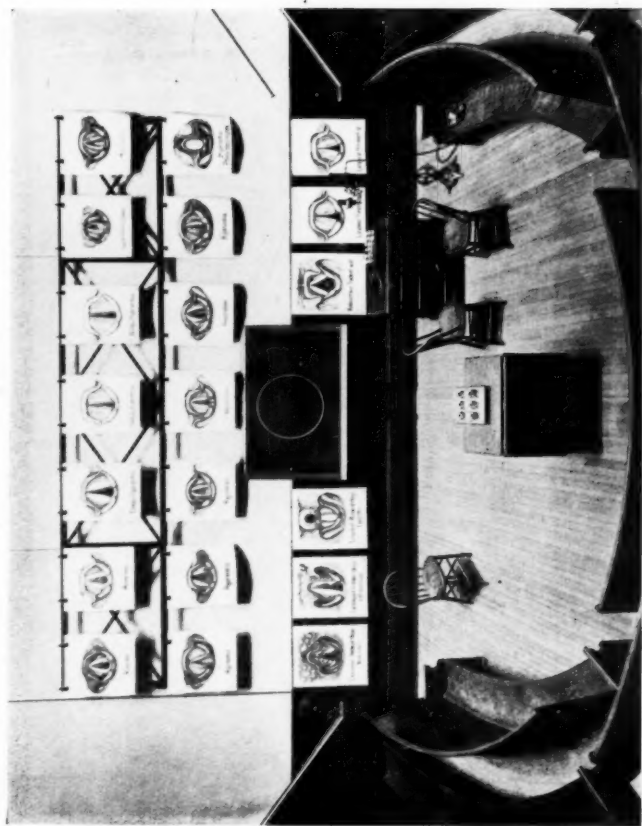
Lecture No. IV.—LARYNGEAL ANEMIA AND HYPEREMIA.
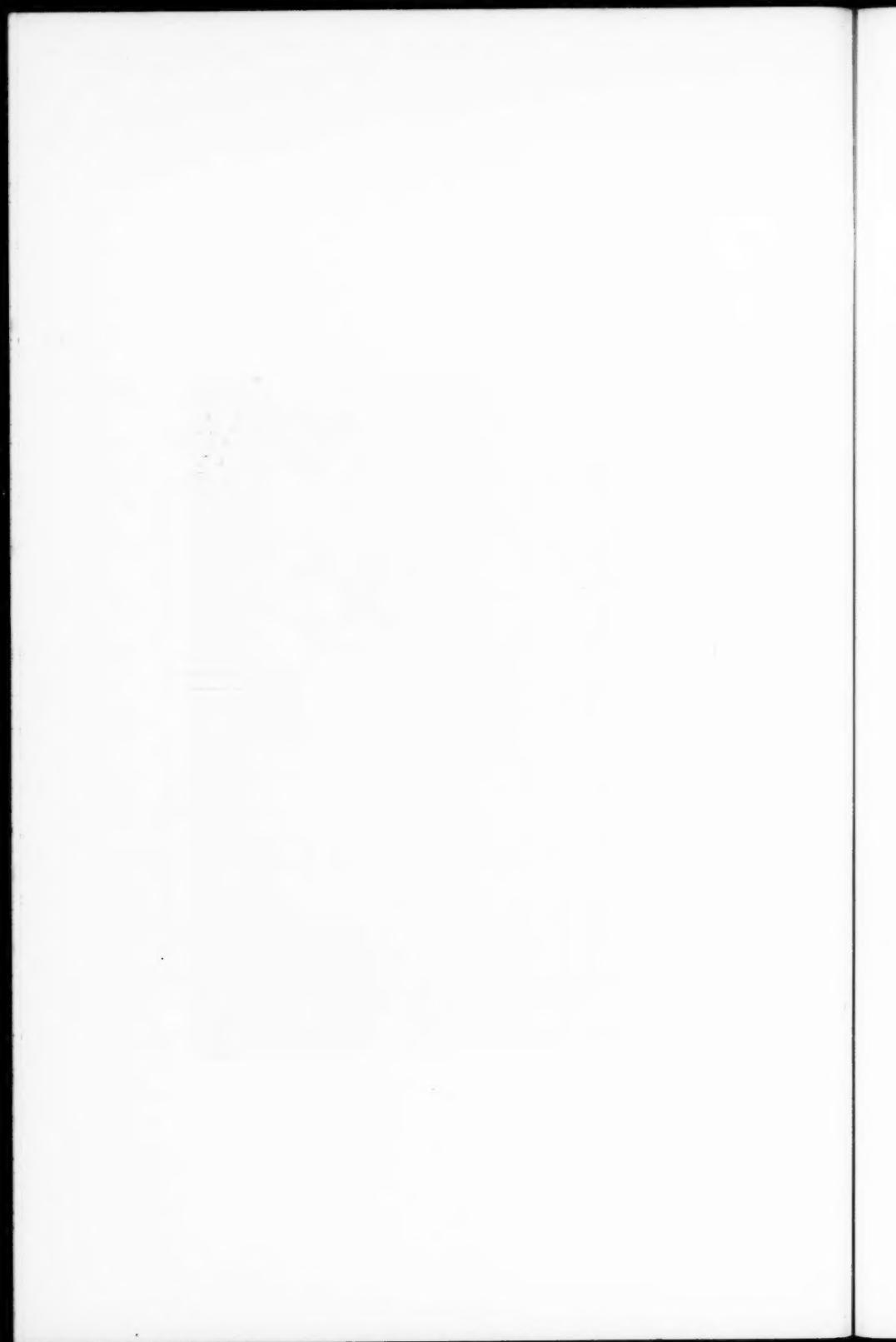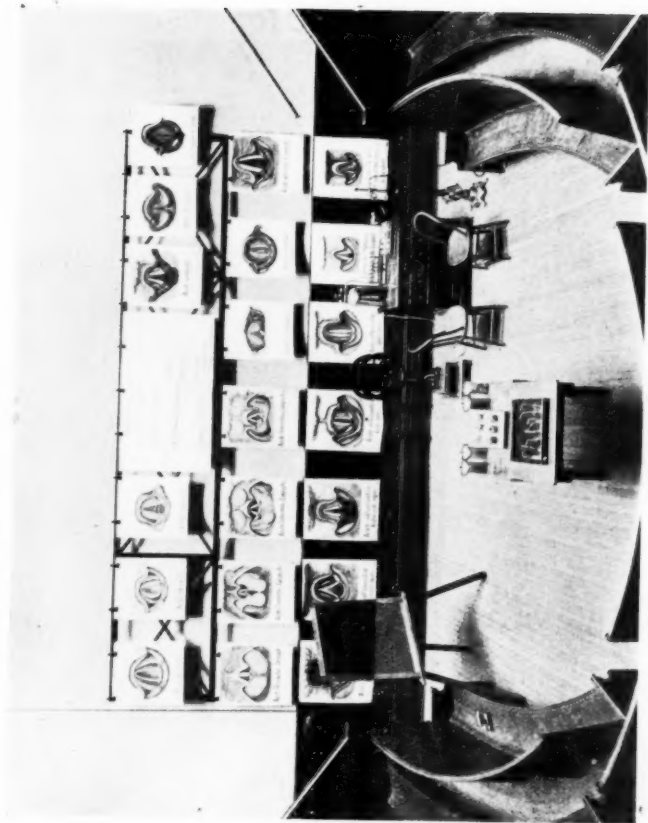
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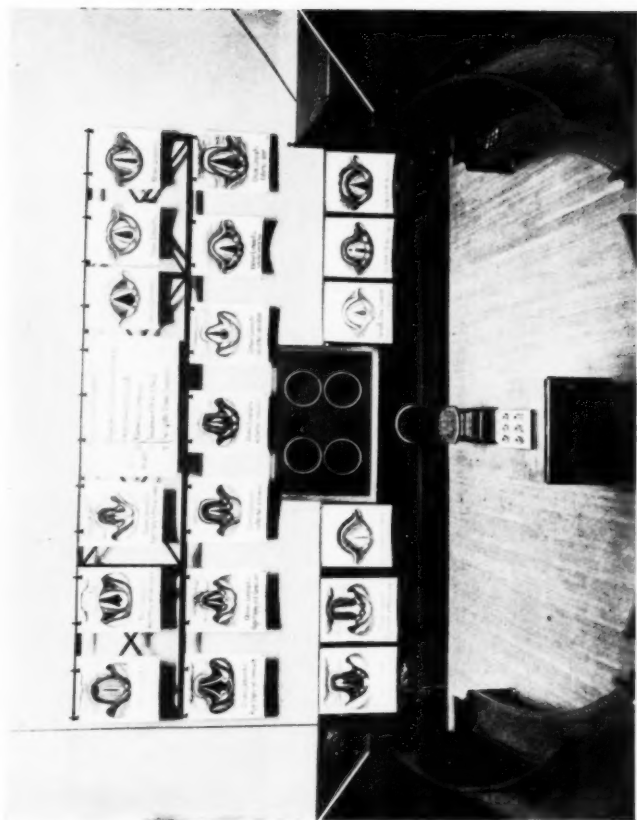
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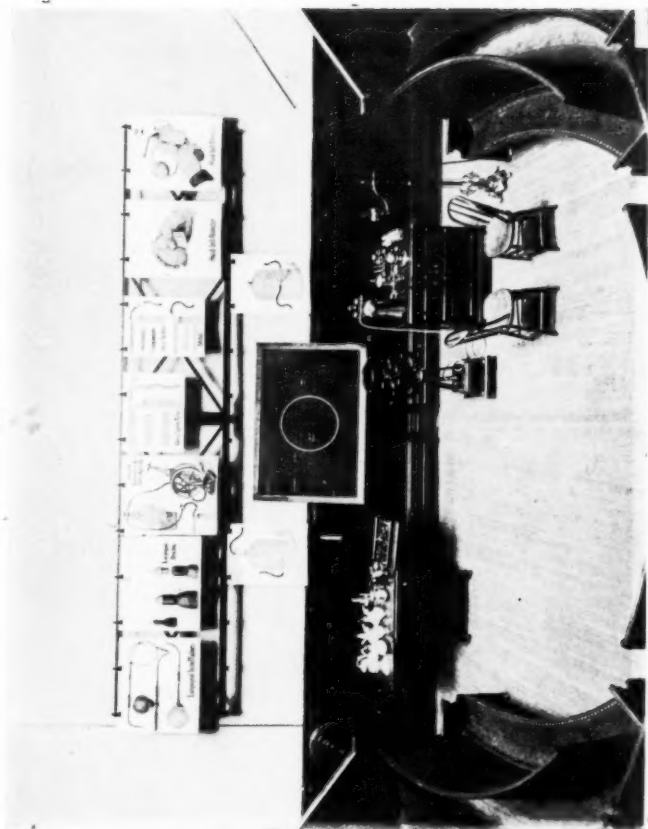
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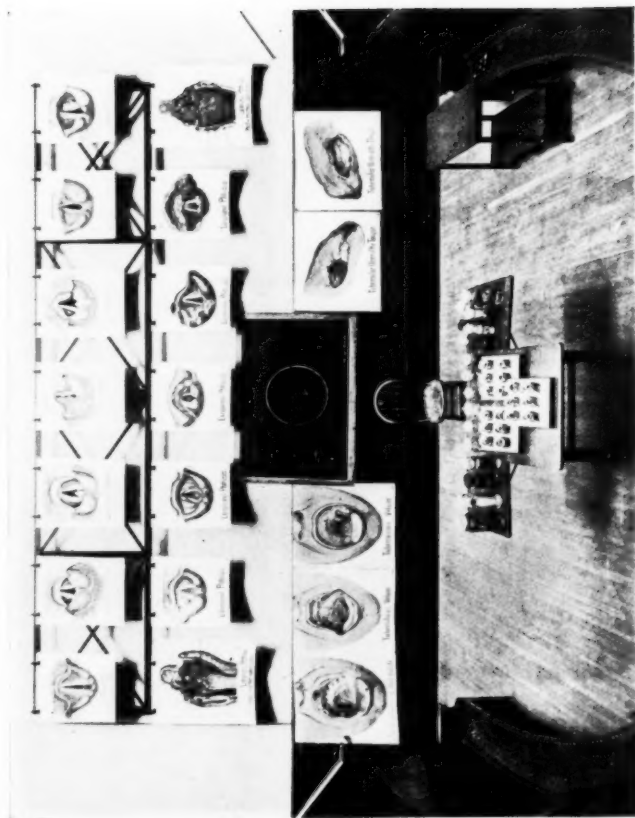
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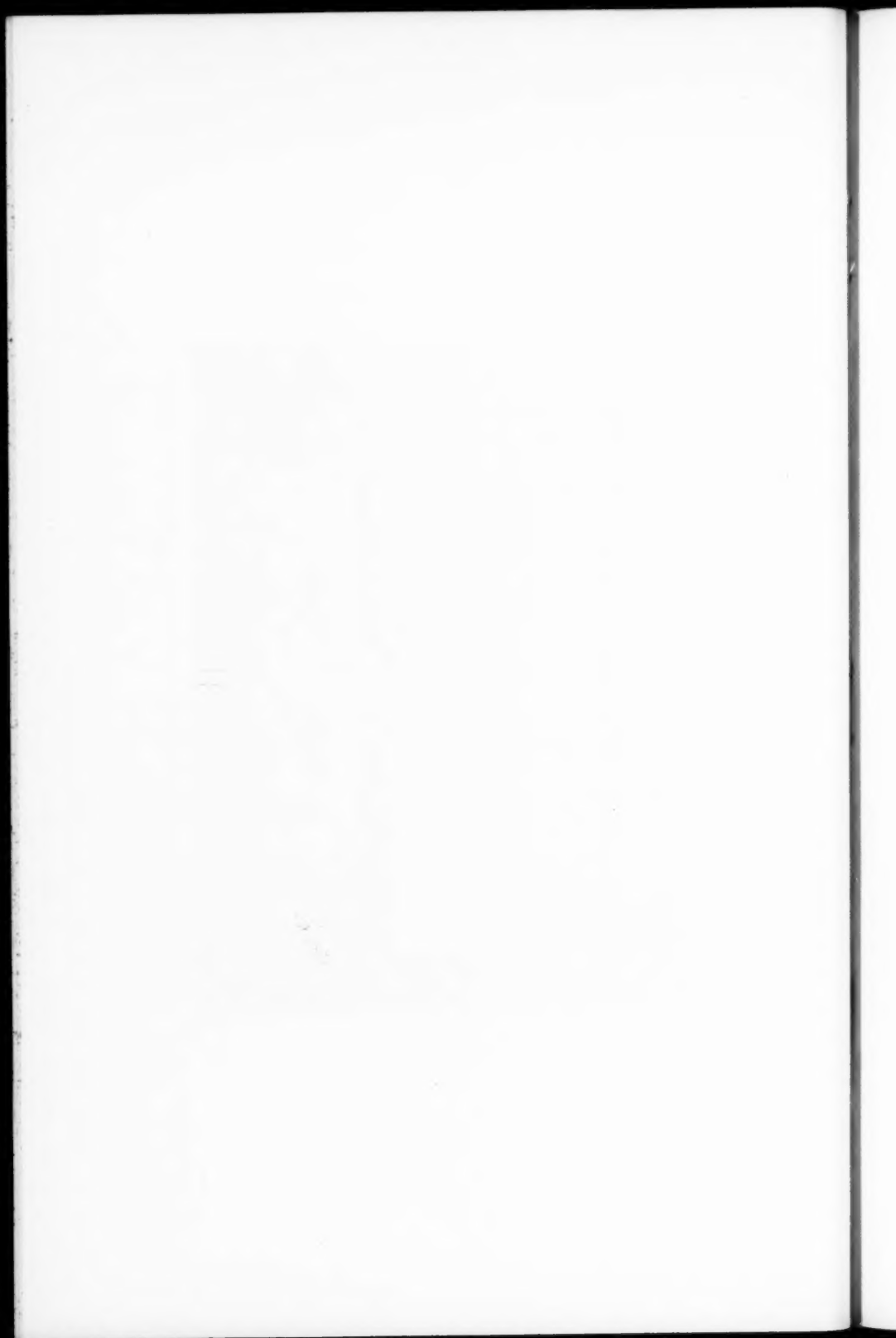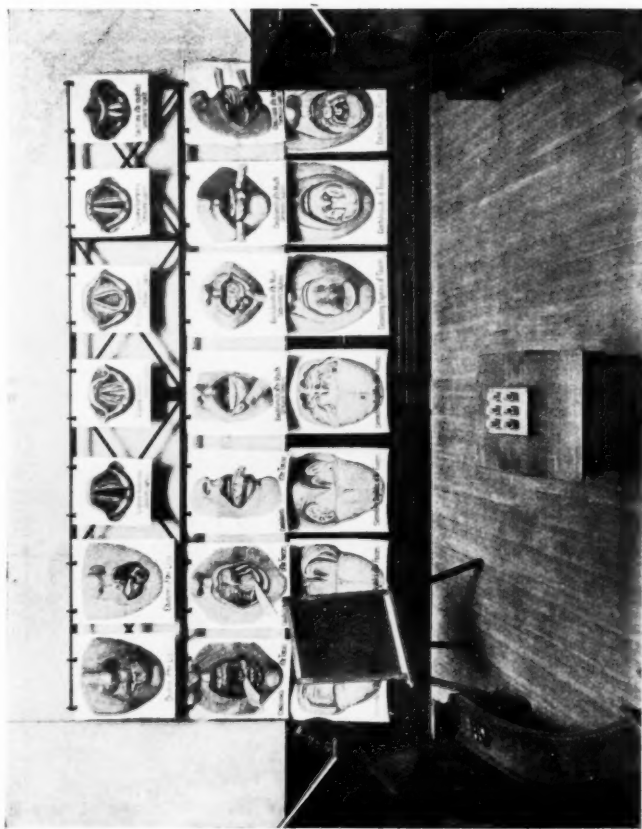
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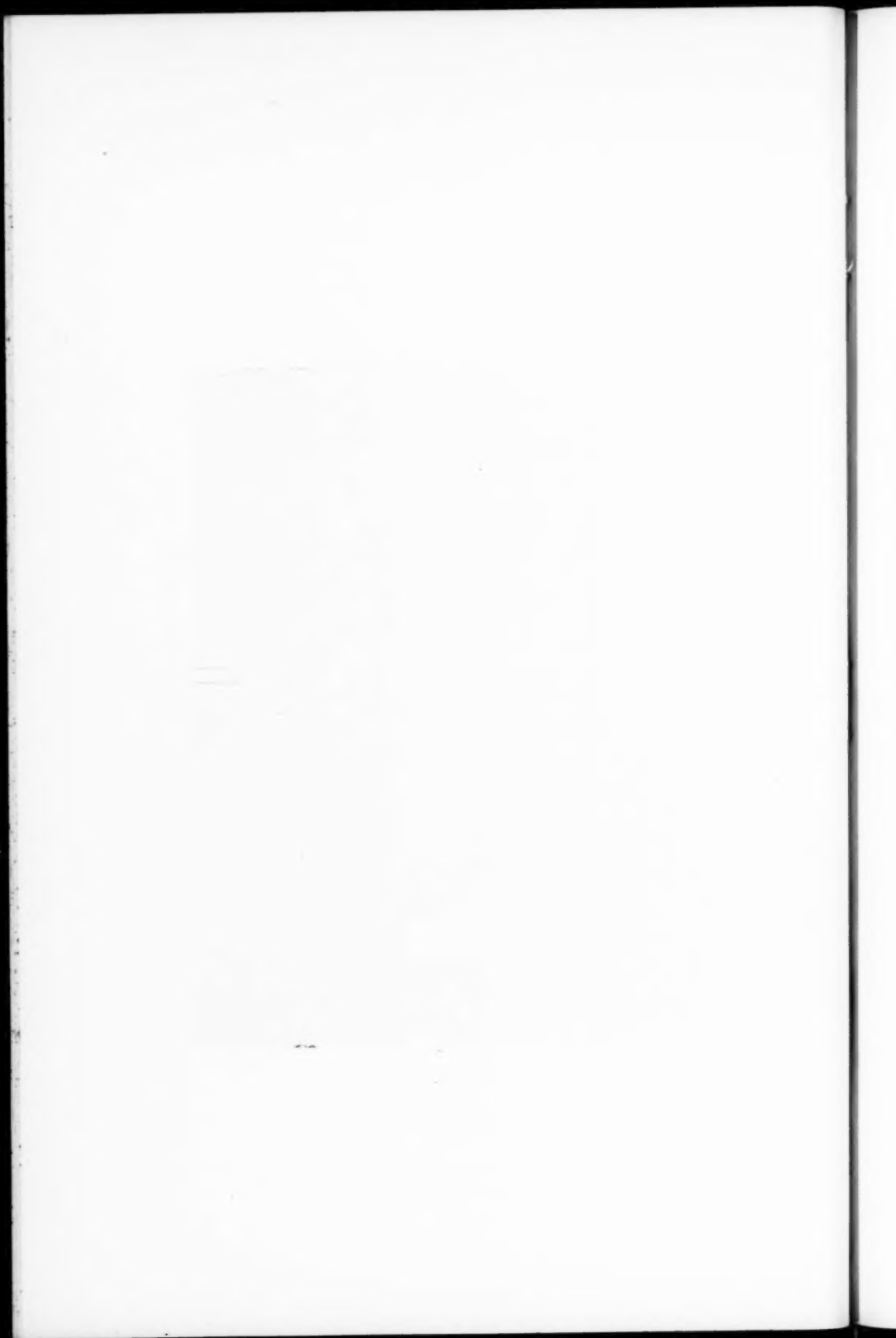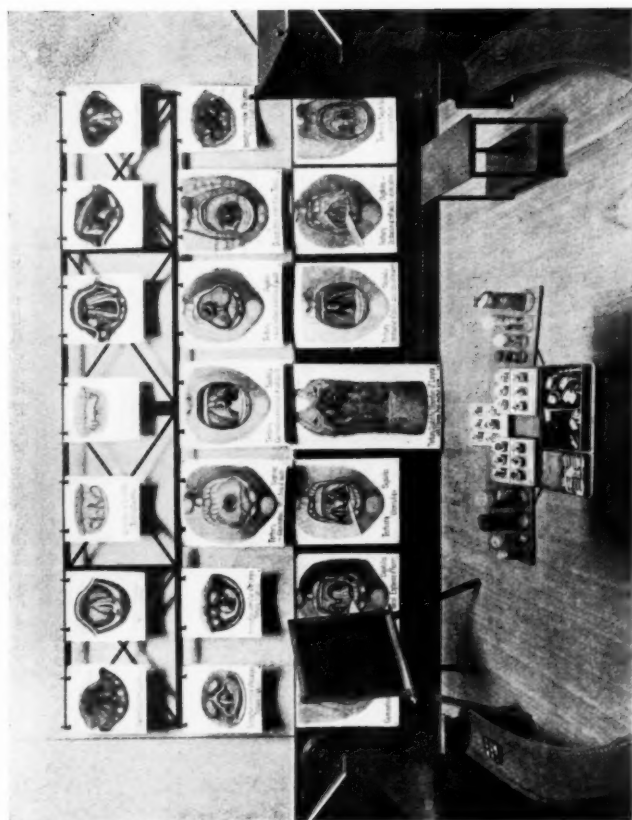
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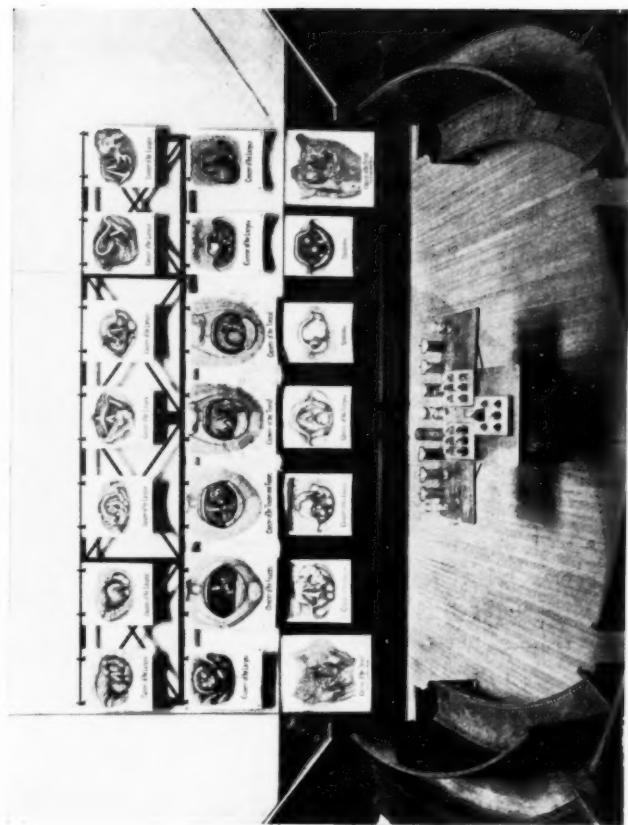
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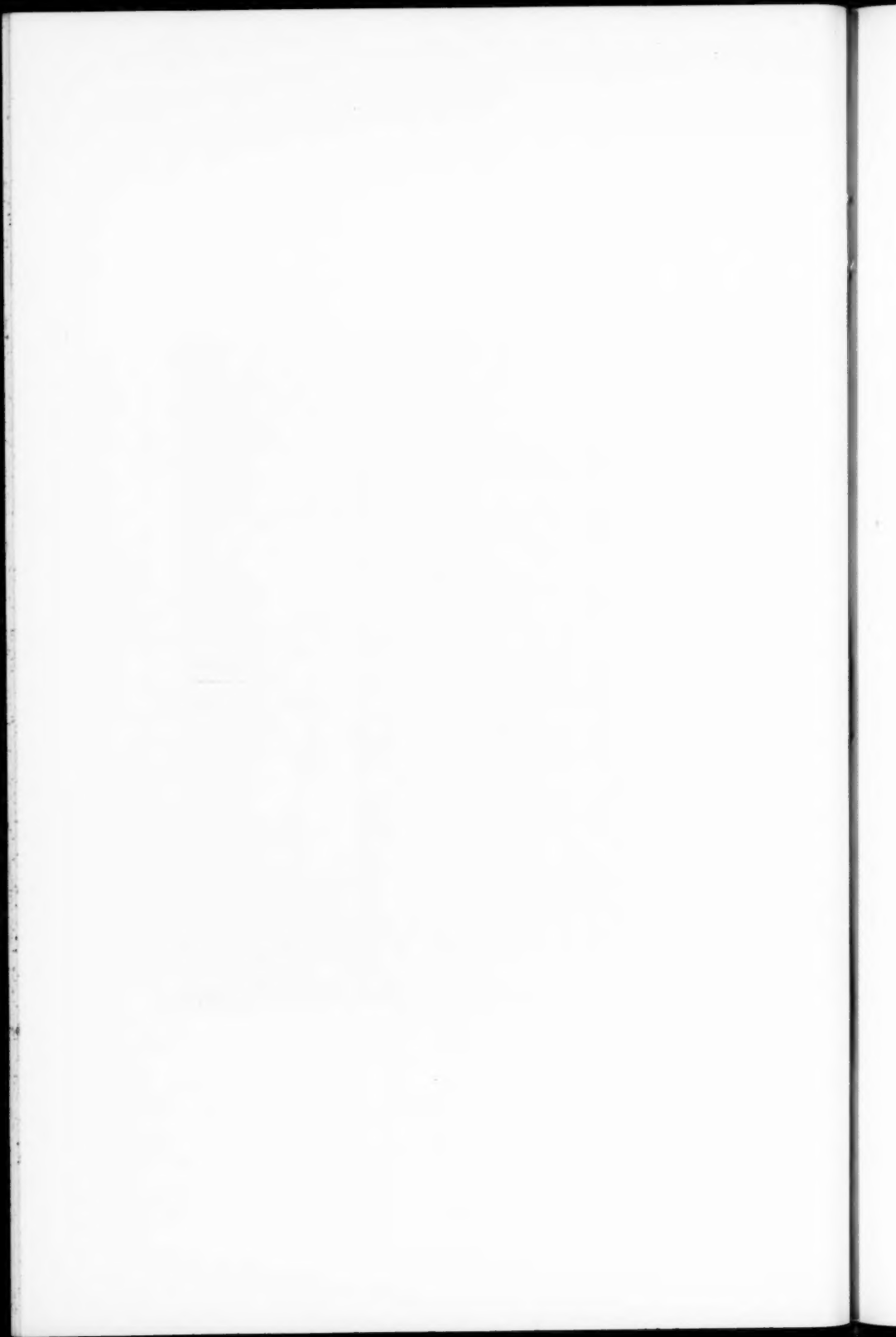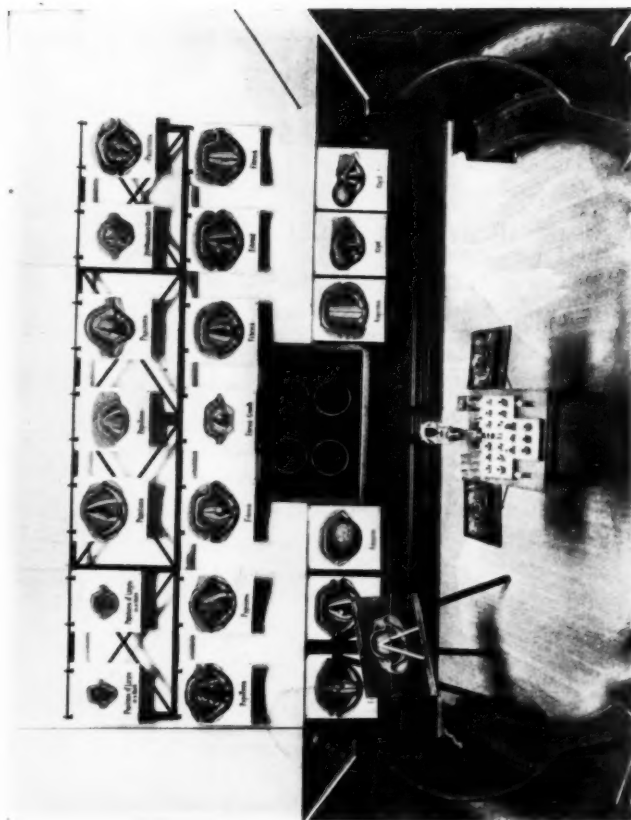
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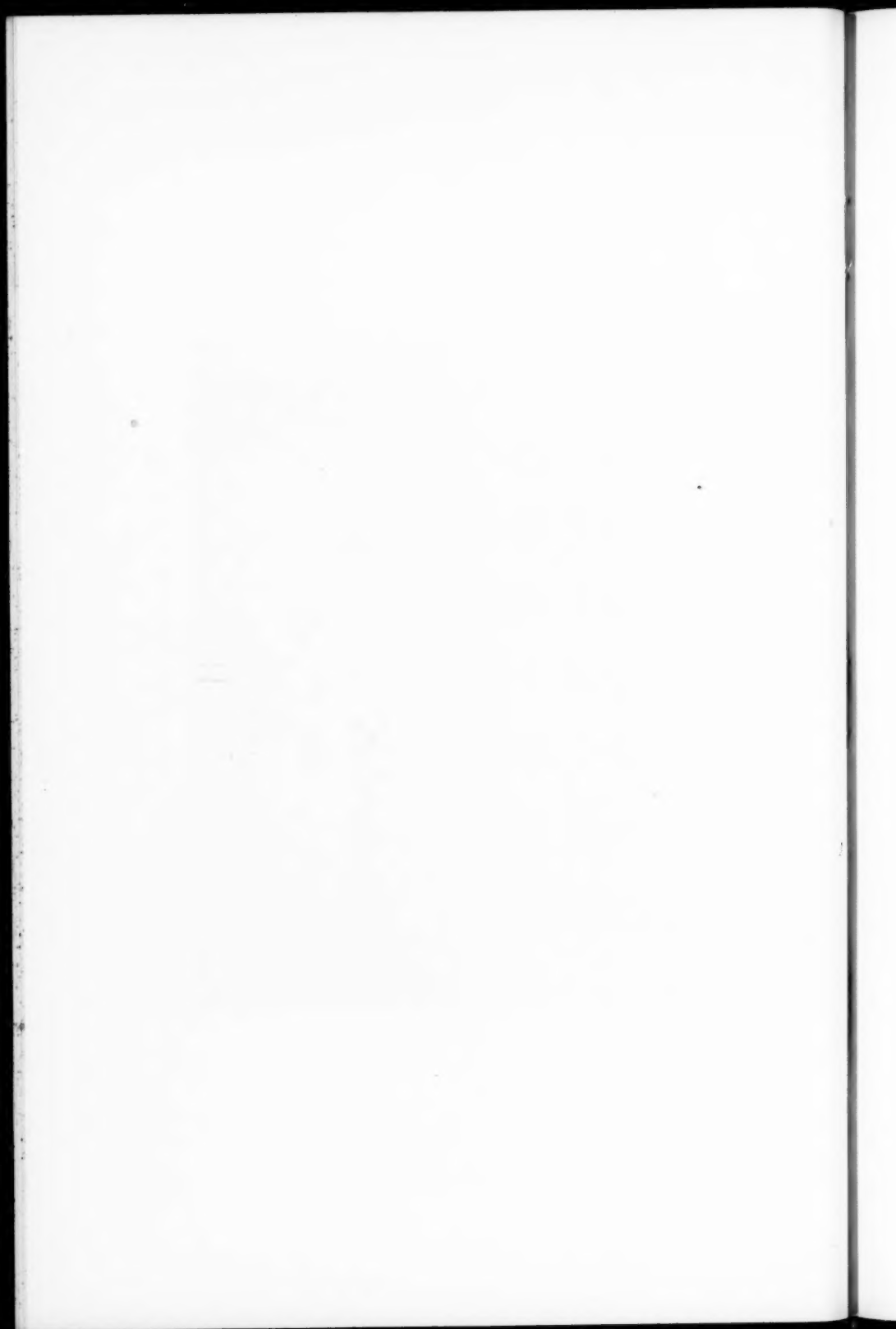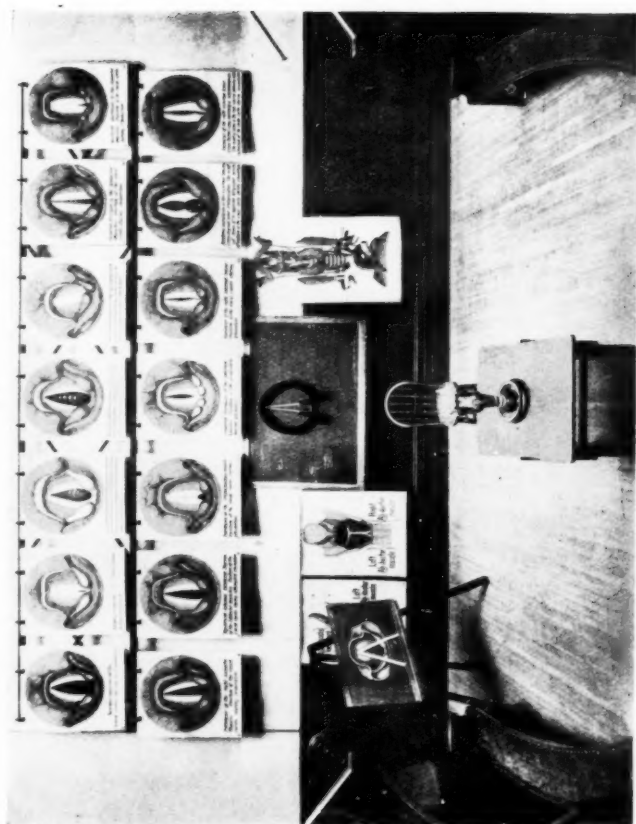
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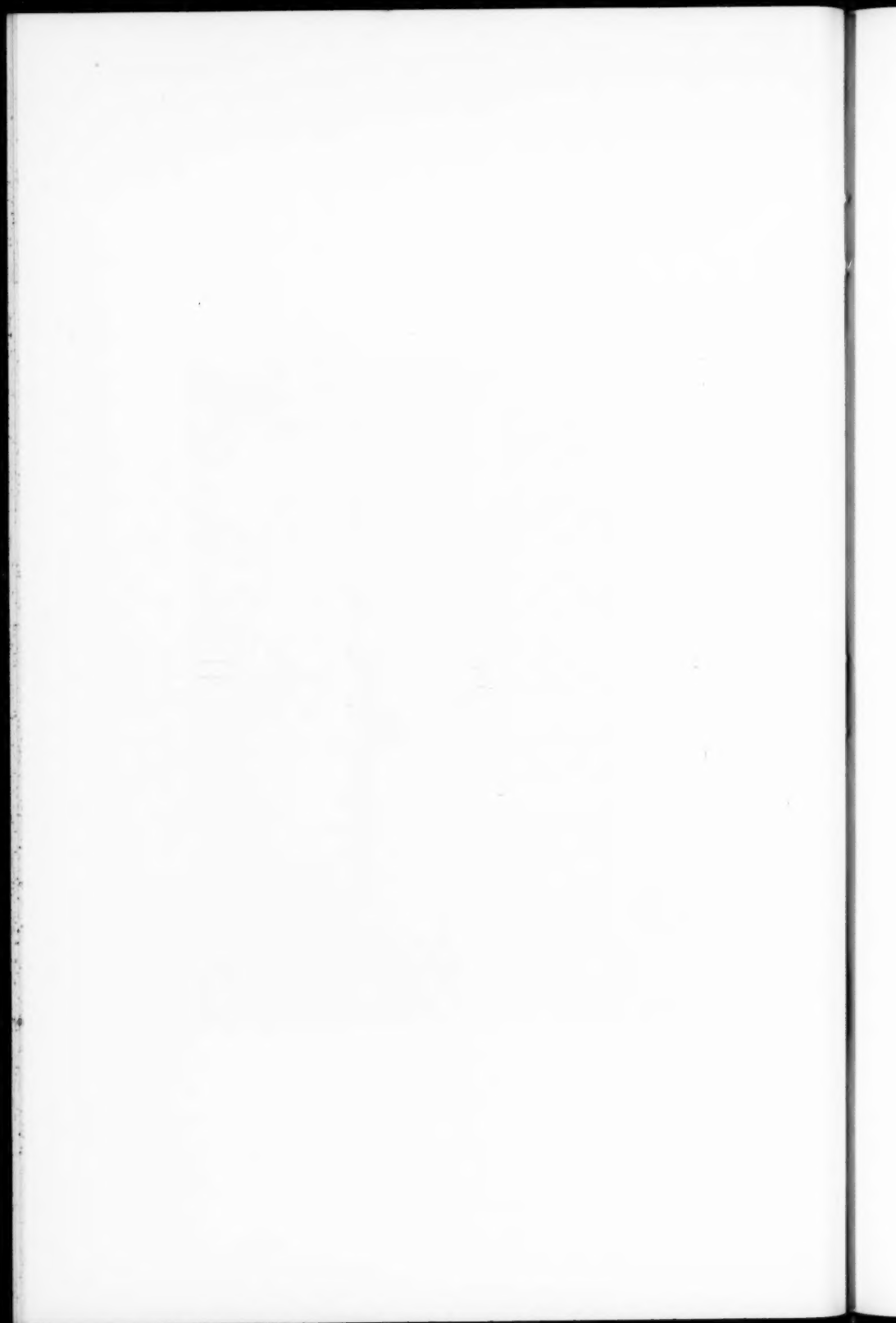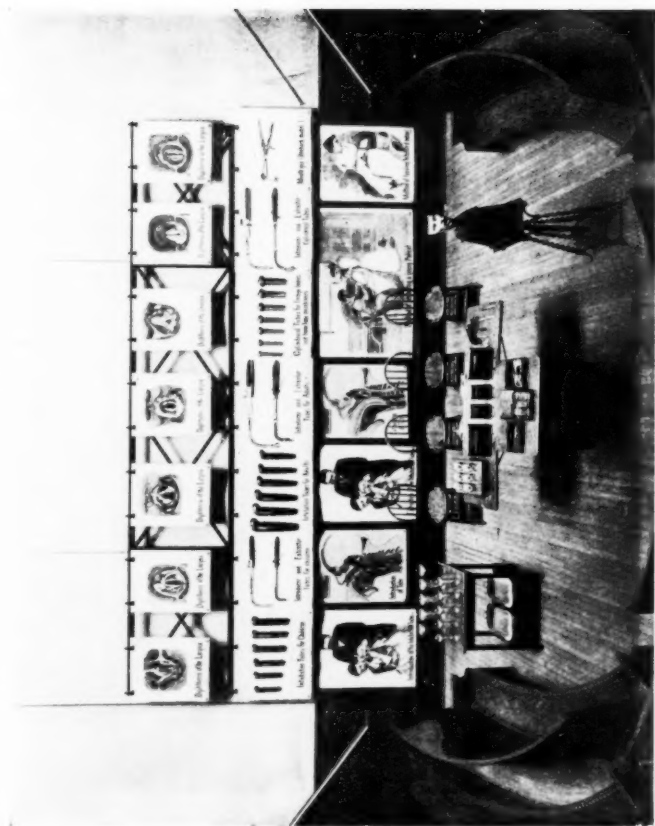
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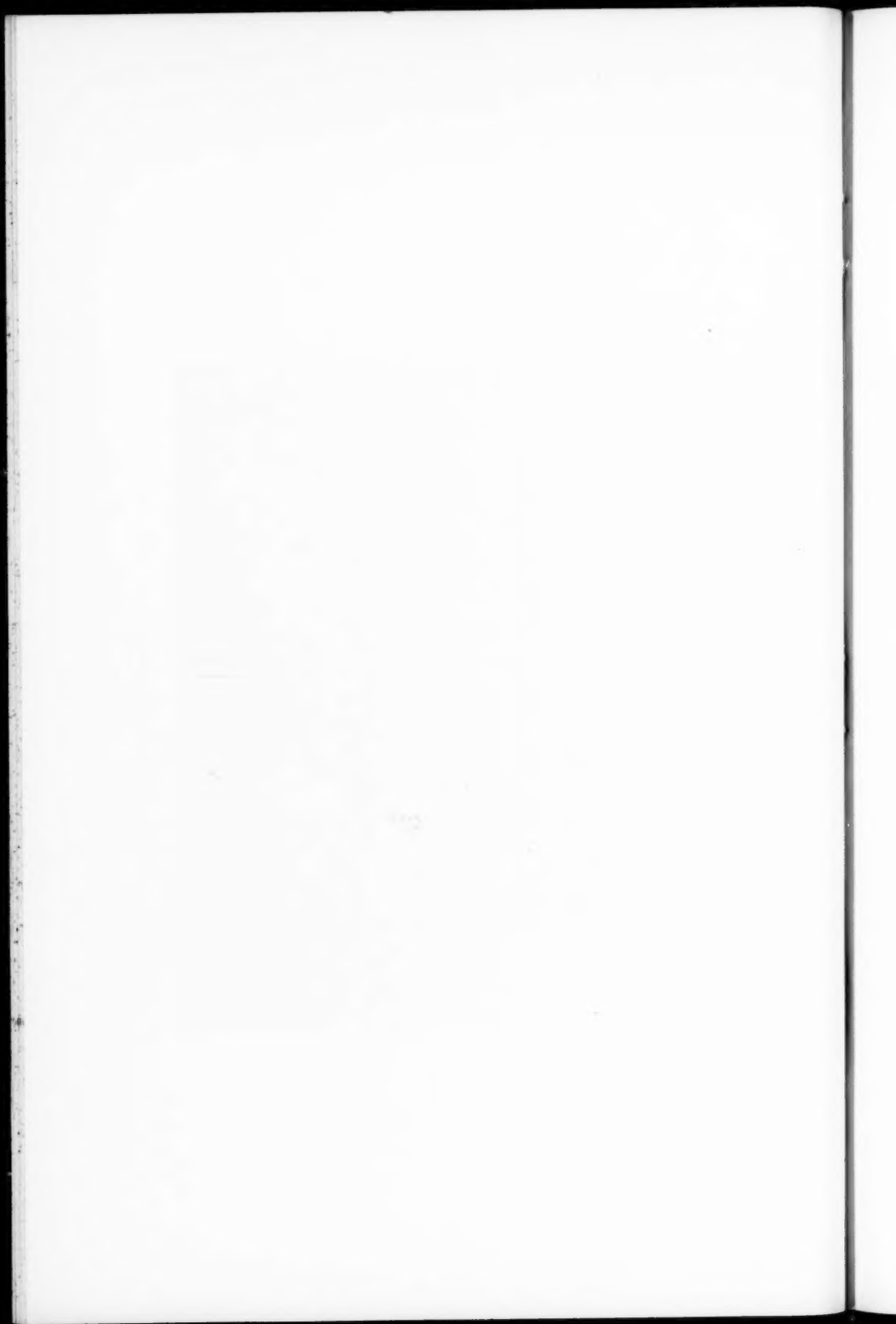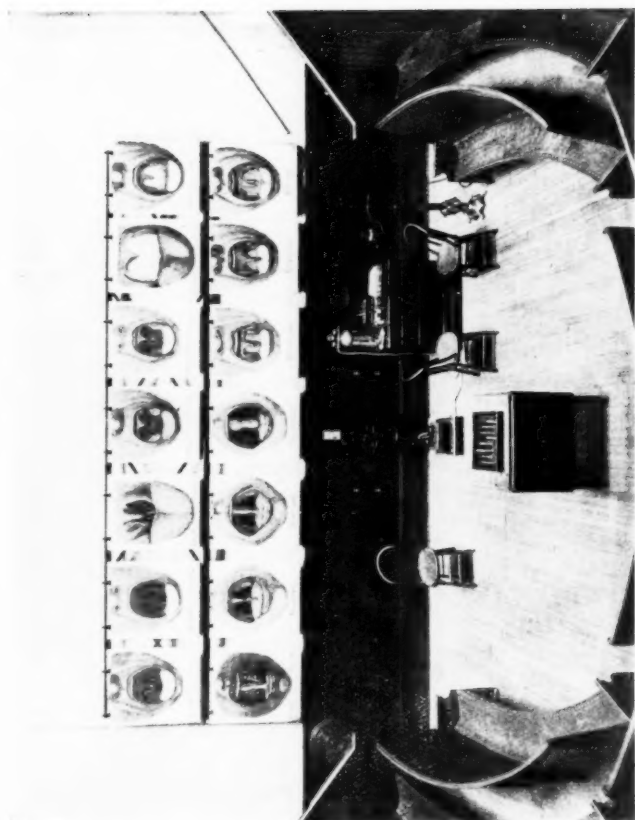
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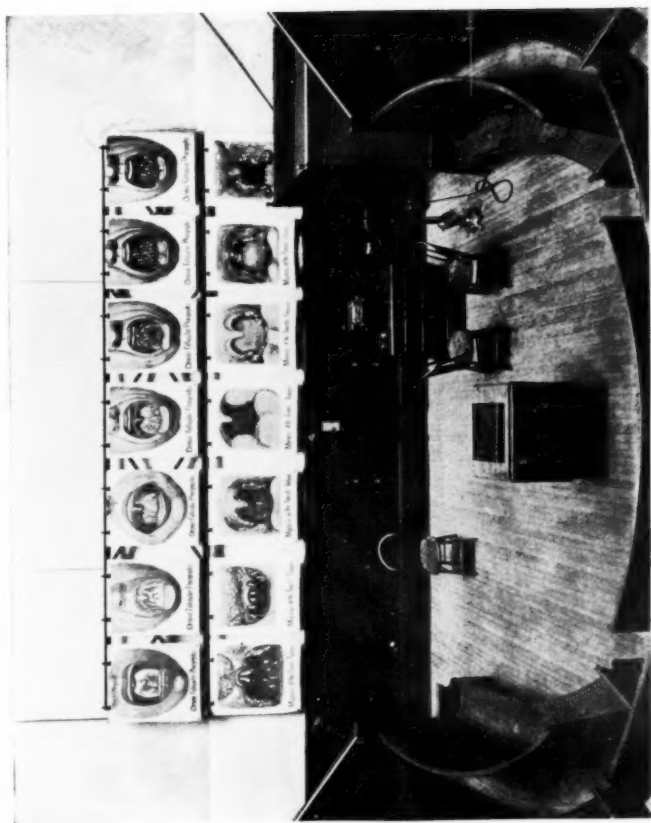
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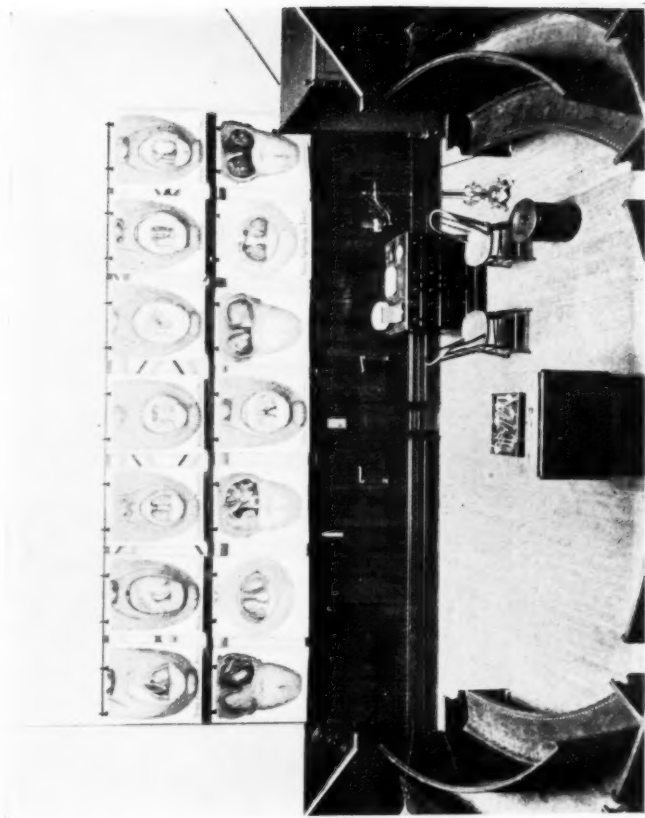
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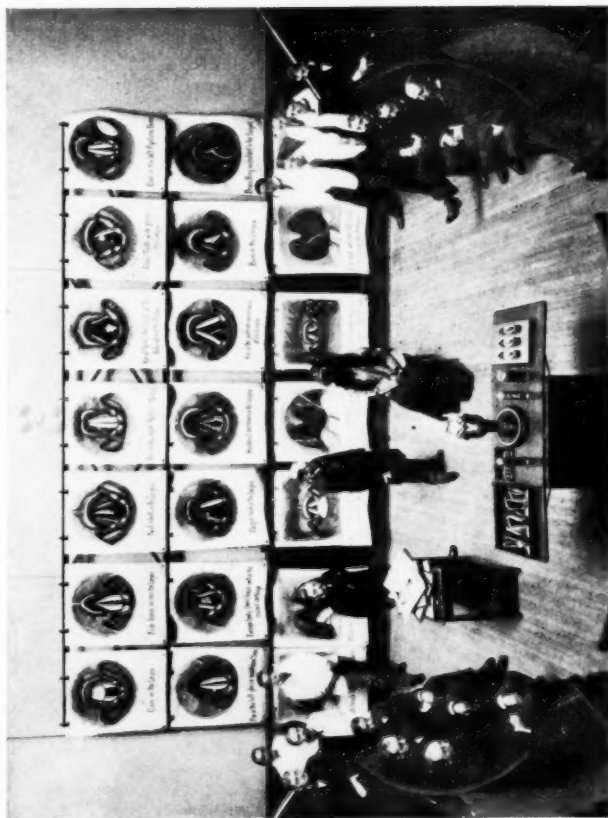
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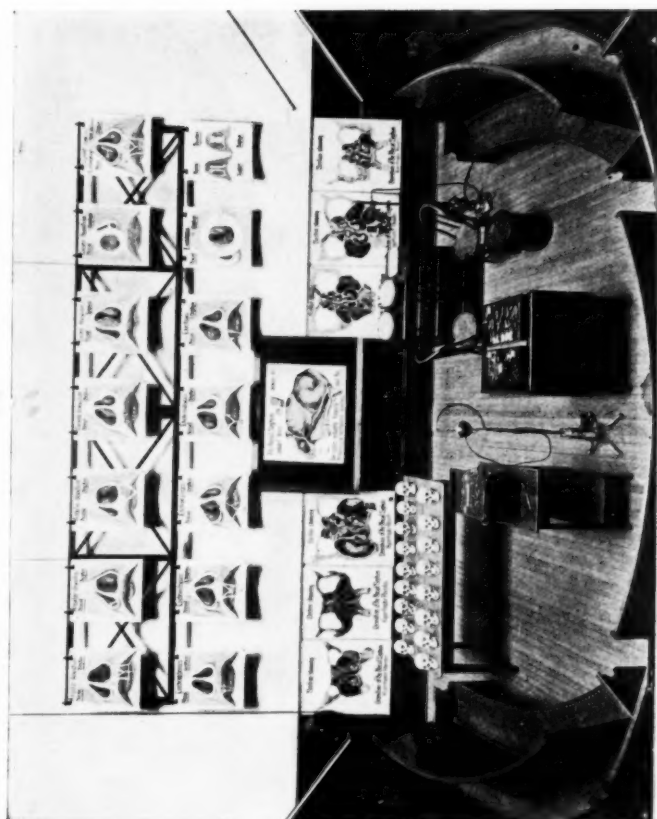
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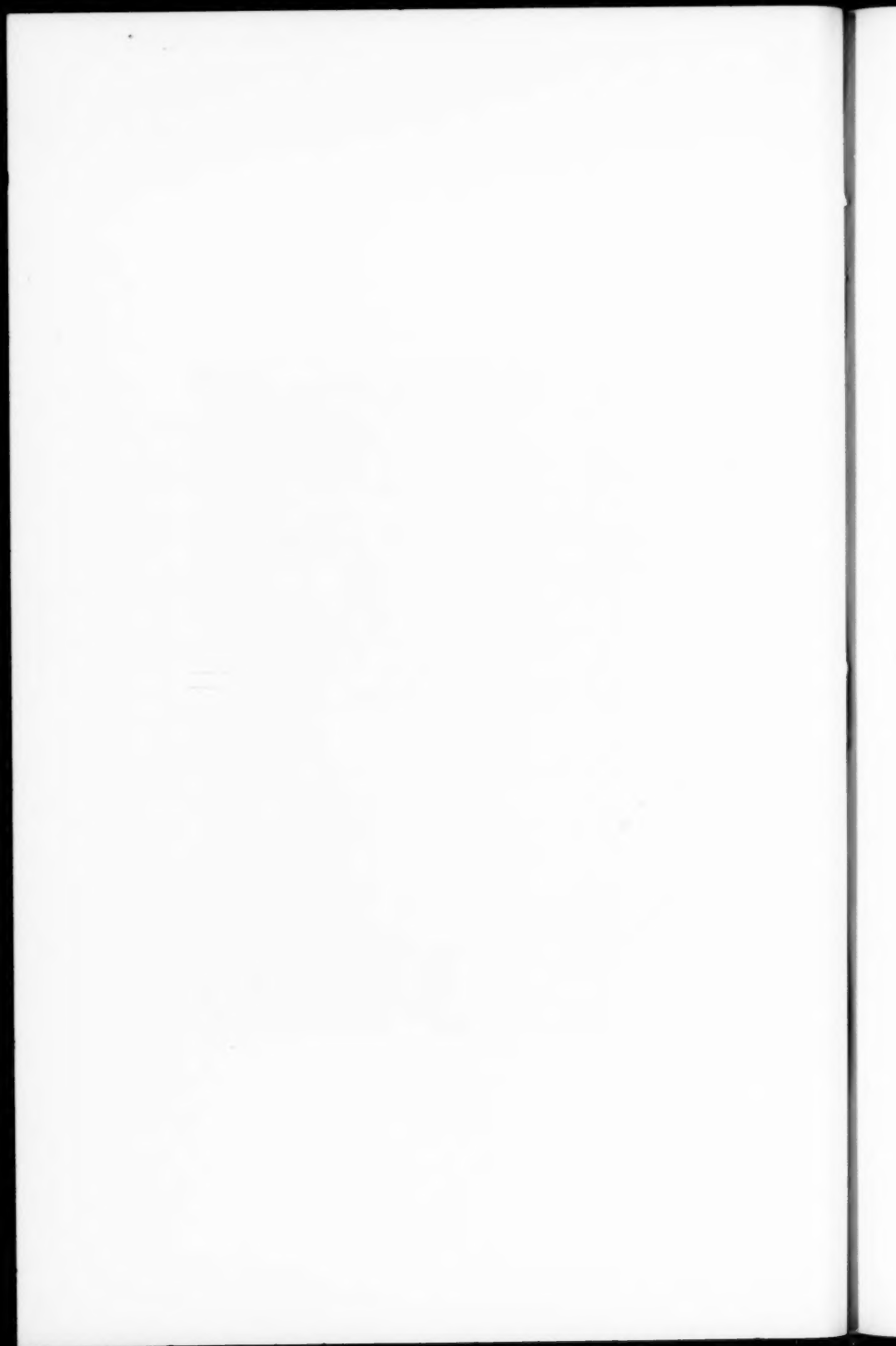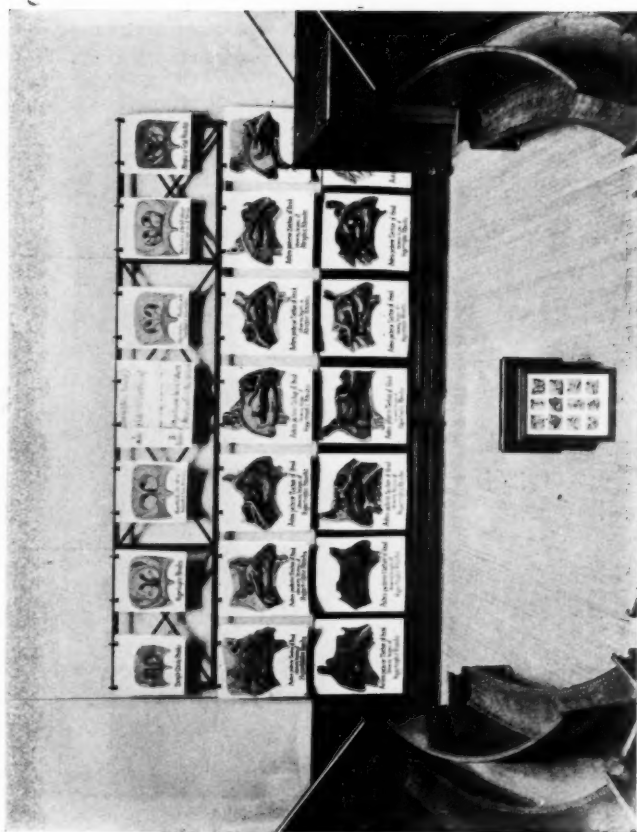
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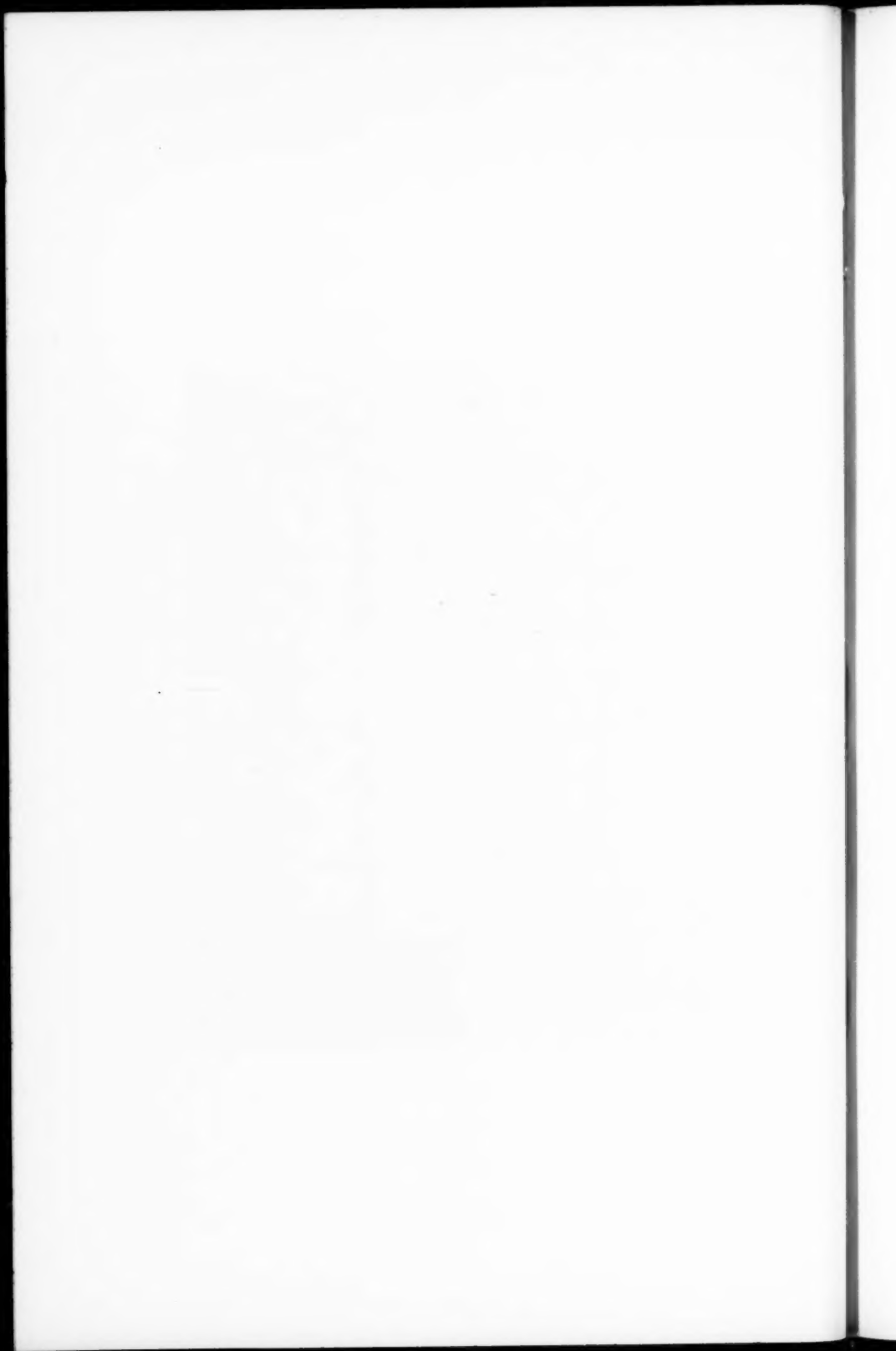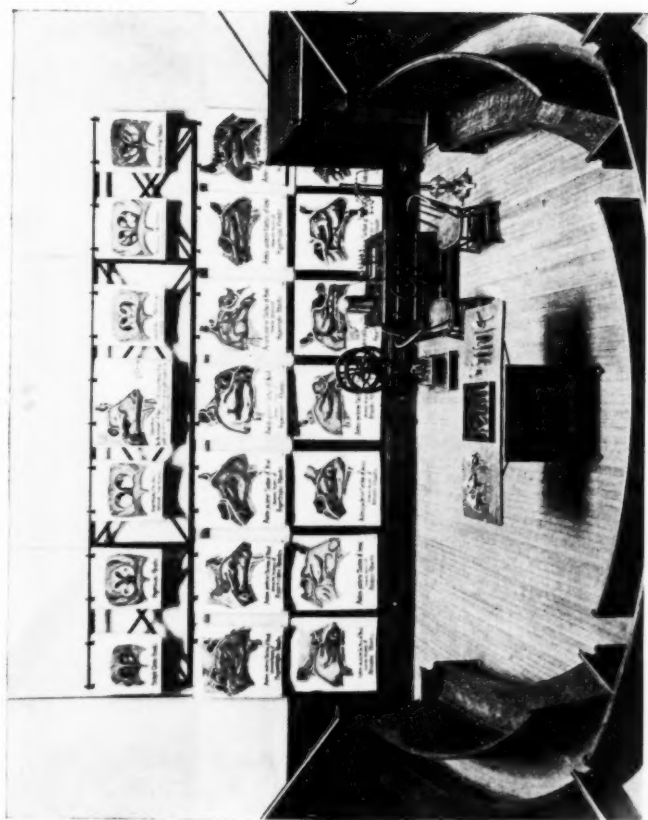
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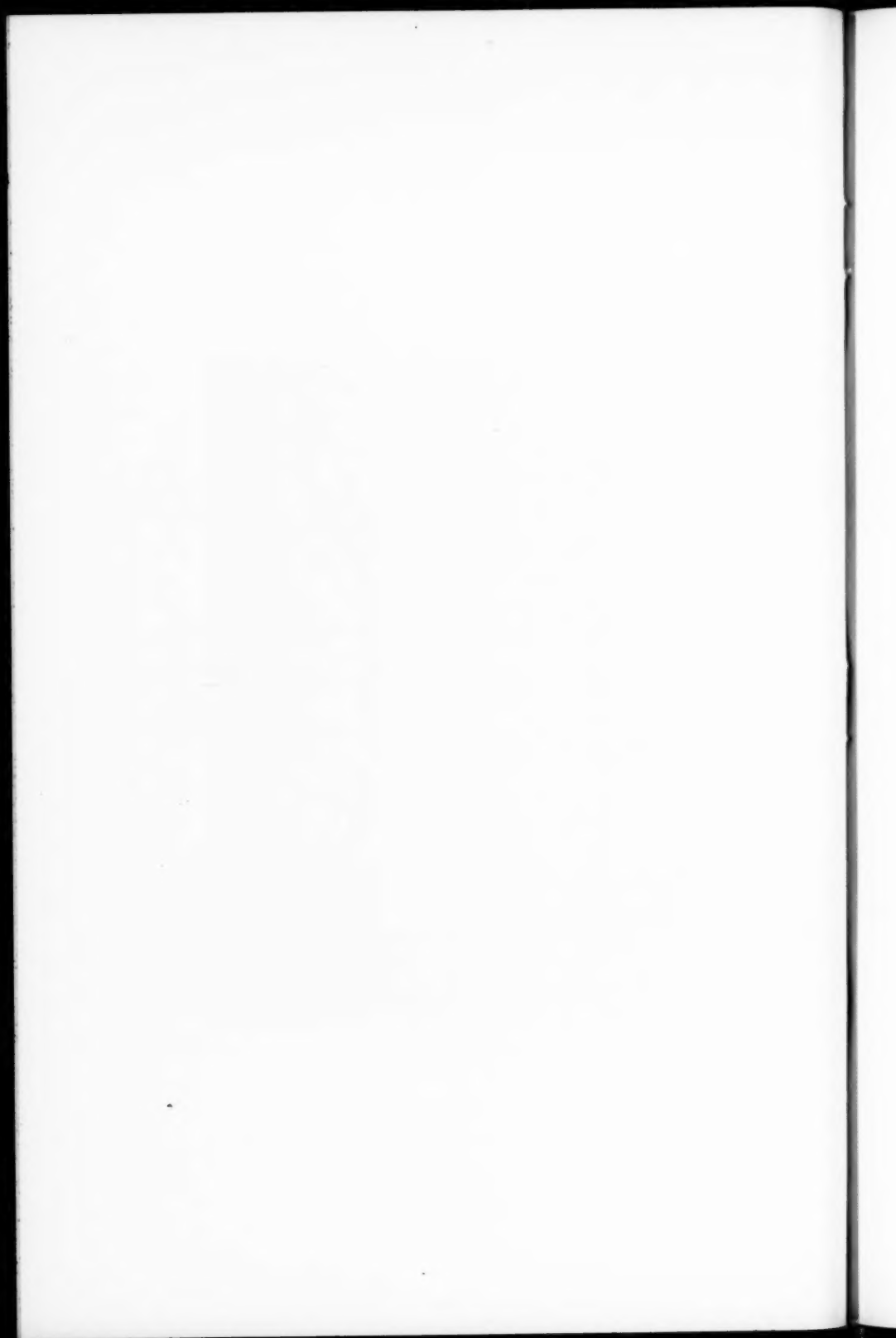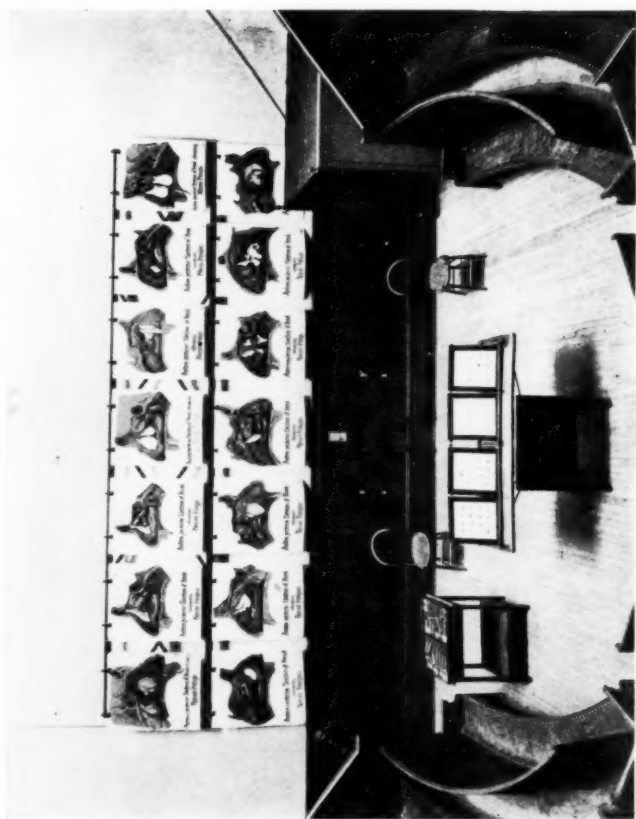
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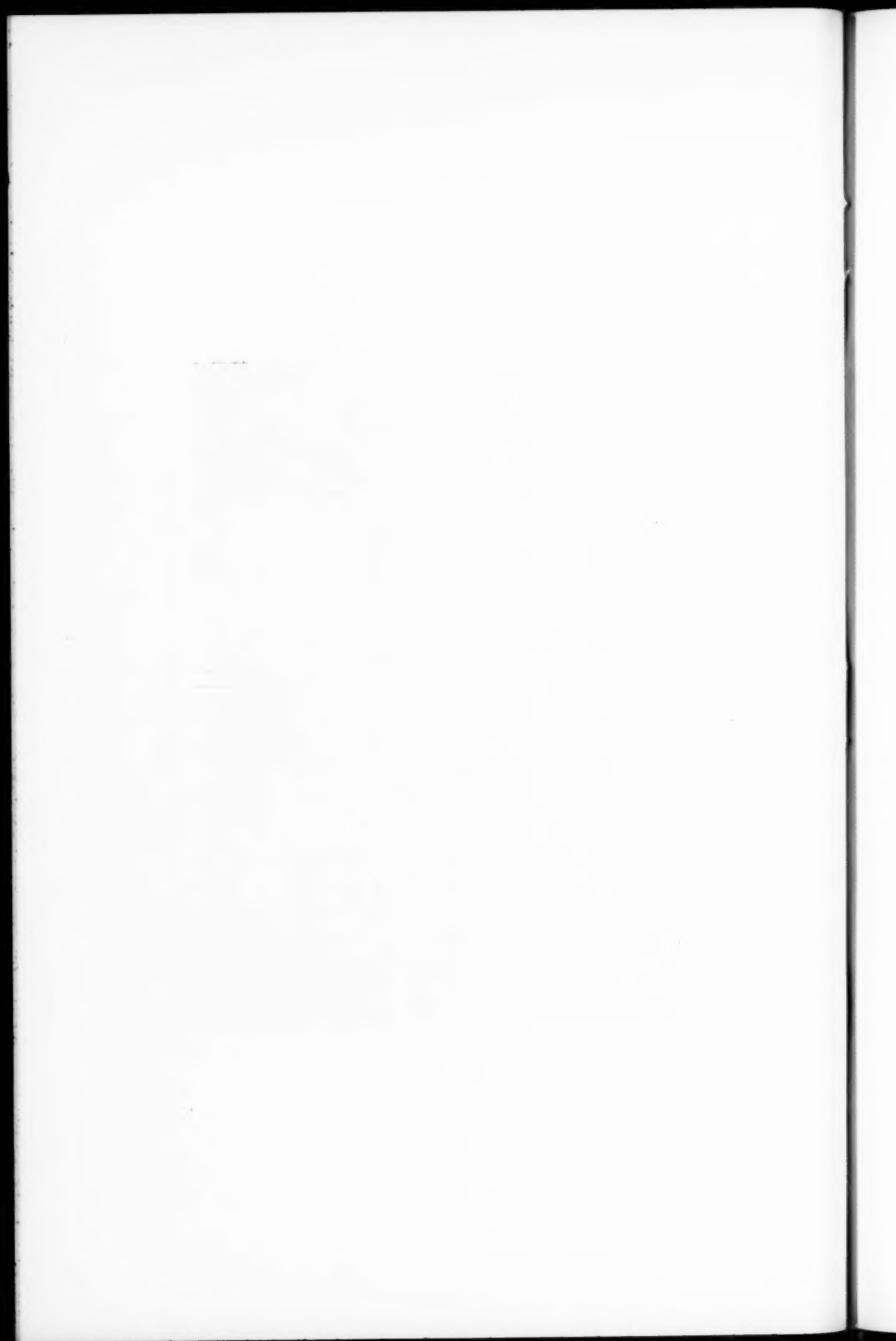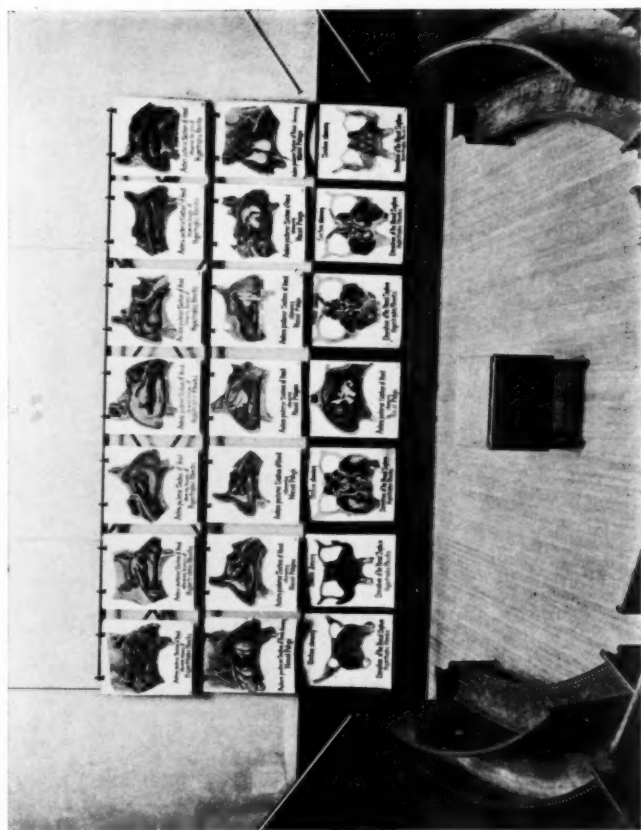
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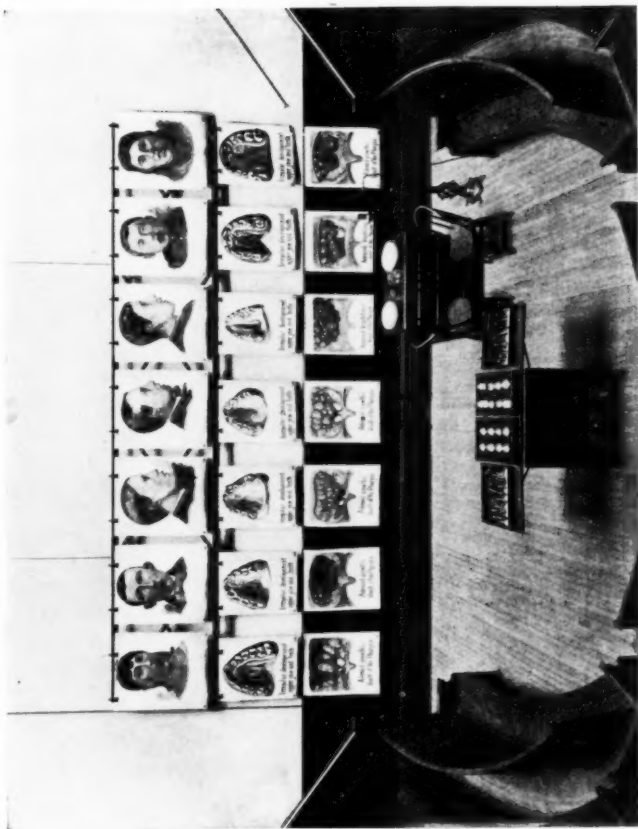
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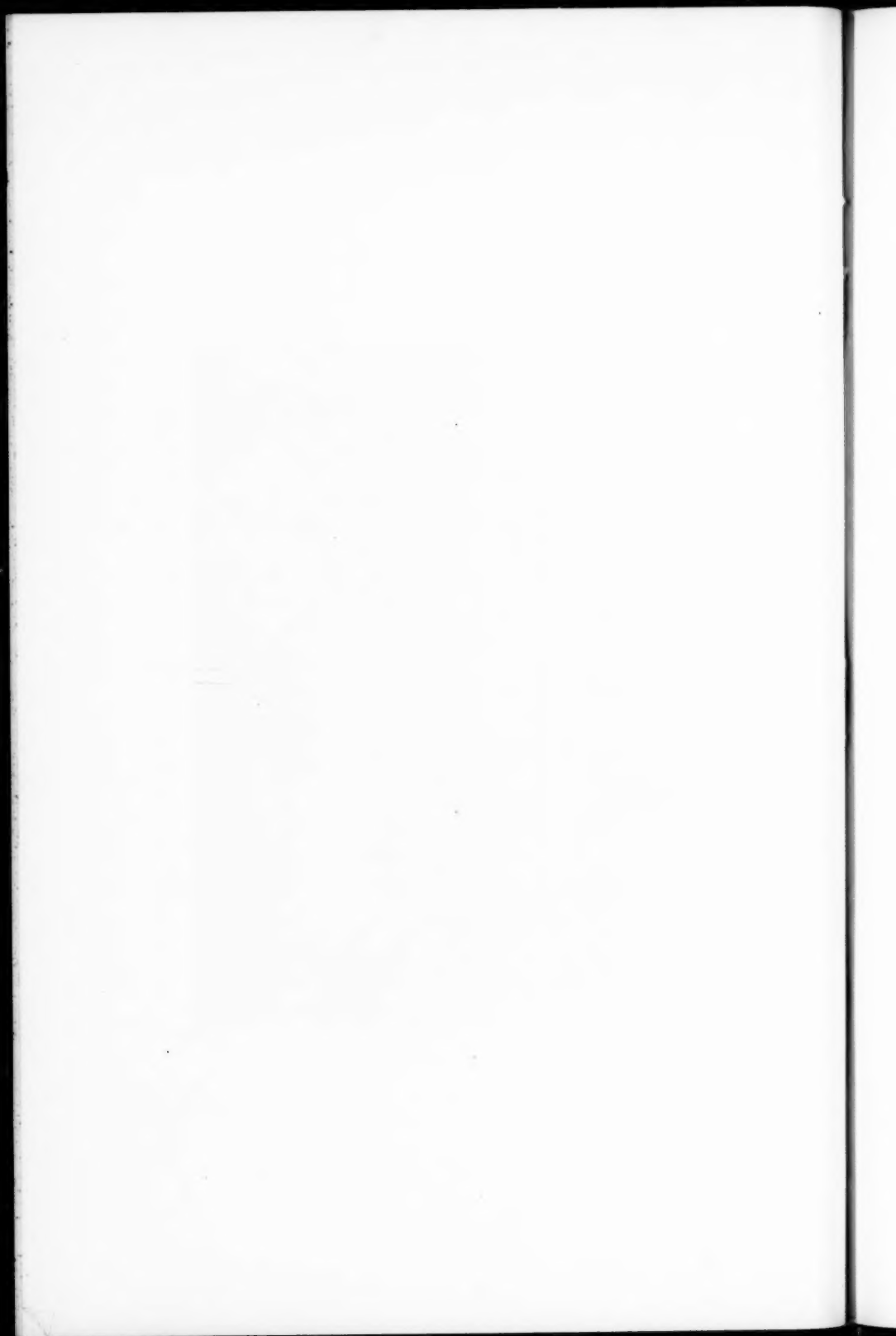
II.

THE INCIDENCE AND PATHOGENESIS OF TONSILLAR CONCRETIONS.*

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Although a very bulky literature upon the subject of tonsillar concretions has now accumulated, very little has been written upon the actual incidence of this condition and no thorough microscopic study of the genesis of tonsilloliths has appeared. A very large majority of the one hundred or more articles on this subject, about sixty of which are relatively easily accessible, are concerned chiefly with the large size of the particular calculus forming the subject of report, each succeeding writer seeming to consider his case worthy of record only in so far as his calculus in grains or grâms outdistanced all others of which he had knowledge. Nor is this surprising in view of the remarkable size occasionally attained by these concretions. A calculus of 85 grains (Butler[8]), 9.5 grams (Rosenberg[52]), 147 grains (Mabon[44]), 189 grains (Anders[1]), 24 grams (Lange[36]) or 26.8 grams (Robertson[51]), to mention but a few, developing within the substance of the faucial tonsil, is quite worthy of description from the standpoint of size alone. Moreover, stones large enough to be detected macroscopically are sufficiently rare to encourage placing them upon record in the form of case reports. On the other hand, calculi of microscopic size can be found very frequently in routine examination of the tonsil by histopathologic methods, but have received practically no attention hitherto.

Of historical interest is the early citation of Lang† (1560) of a case of his own and also of one by Nicolas Blégny. In a critical survey of the early literature Terrillon[59] has shown

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how few additional cases were added by the references usually
cited as following Lang in chronologic order, for the two of
Kentmann and Schenk refer again to the one of Lang,
although frequently counted as new cases. The instances re-
ported by Donatus in 1597 and Rivière in 1688 were not based
on first hand knowledge. Hoffman's case, reported in 1717,
is considered by Terrillon[59] to be a true addition to the list and
also those of Bailheron, Bourguet and Souque, discussed by
Louis in 1774, although these observations covered a period
of thirty years or more preceding that date.

In the nineteenth century the first additional cases were the
three of Wilson, reported by Monro[44] in 1811, two of these
patients being members of the same family. The further ac-
cumulation of case reports during this period need not be
detailed here, as this ground has been sufficiently covered by
others. References are given in the appended bibliography.

A survey of the more recent literature shows that tonsillo-
lithiasis has been reported by more than thirty-five writers
since 1900. In 1901, Kayser[34] reported a calculus measuring
3.5 by 2.8 by 1.6 cm., occurring in the upper pole of the left
tonsil of a twelve year old boy. Other cases were reported by
Jacques[32] and by Guarnaccia.[27] Gray's[26] case in the same year
occurred in a man 68 years of age who had chalky deposits
in the phalangeal joints. Tonsillar calculi developed in both
tonsils and were discharged spontaneously. The article by
Feigin,[20] in 1903, is not readily accessible. The large calculus
described by Mabon,[41] in 1904, was expectorated by a 54 year
old male who had had sore throat for a period of three months.
The site of development was represented by a cavity in the
right tonsil. In this year, also, Compaired[12] briefly reviewed
the earlier literature and reported an instance occurring in
a woman aged 43. He recognized the crypt origin of tonsillar
calculi and laid stress upon the calculous diathesis as favoring
concretion formation in organic material of any sort in the
crypts. Then follow Jackson[31] and Girou,[25] in 1905; Struy-
ken[56] and de Simoni[17] in 1906, and Scheven[54] with five calculi
removed from the superior tonsillar fossa of a fifteen year old
boy in 1907. Of these he has good photographic illustrations.
Earlier in this year Miodowski[43] had described and illustrated
an early stage of the deposit of lime salts in the interior of old

actinomyces-like colonies in the tonsillar crypts. He seems not to have associated this process with the origin of larger calculi. Also in 1907 appeared the articles of Landa[35] and Morroway,[45] the latter reporting a concretion from the pharyngeal tonsil. Anders,[1] in 1908, described his large calculus, weighing 189 grains and measuring $1\frac{7}{8}$ inches in its greatest diameter, while that reported by Mignon[42] weighed nearly as much. In 1909, three additional reports appeared, those of Finder,[21] Solters[55] and Vargas.[63] The first mentioned assigned the origin of a tonsillolith in a male, 20 years of age, to lime salt deposit in the inspissated pus of a peritonsillar abscess. Trautmann[61] discussed in 1910 the relationship of plugs of desquamated epithelium in the tonsillar crypts to the formation of calculi. In the same year Parker,[49] Dupond[19] and García e Hurre[22] each added new case reports.

In the first year of the following decade Delobel[16] and McCarthy[40] each reported tonsilloliths of large size, the one removed by the latter with "ovum forceps" measuring 2.5 by 2.5 by 1.5 cm. and weighing 4.635 grams. The paper of Turskiy[62] in 1912 is not accessible to me. Maclachlan[39] briefly described the crypt origin of calculi in the course of his monograph on "Tonsillitis," published during that year. This was followed by Bertrán y Castillo[4] in 1913 and Curti[15] in 1914. The calculus described by Hayton[28] in 1917 measured 2.7 by 2.2 by 1.8 cm., weighing 7.9 grams. The case history reported by Swain[58] in 1920 was of unusual interest in that the same tonsil had been four times the site of calcareous accumulation during a period of twenty-nine years. In the same year Müller[46] made use of Roentgen rays in investigating a marked swelling of the left tonsil region. Shadows of two foreign bodies, more dense, even, than those of the teeth, were noted and two calculi, which together weighed 4 grams, were removed by incision.

In 1921 Woodman[66] reported the forceps removal of a recurrent calculus of such size that the anterior pillar was lacerated. This was studied by radiography (after removal) and was found to be very opaque to X-rays. The shadow as illustrated, however, shows definite radial striæ near its periphery.

Suné y Medán,[57] 1921, in reporting a calculus of the left tonsil in a patient forty-five years of age, gives the most com-

plete clinical presentation of this subject that has appeared
in recent years. He adds very little, however, in respect to
the manner of formation and presents no microscopic data.

Summarizing the literature, then, it appears that a fairly
clear cut clinical conception of the condition of calculus of
the tonsil has been established. It is evident that tonsilloliths
of significant size are comparatively rare, that they occur much
more frequently in adults approaching middle life than in chil-
dren, and that at times they reach an extraordinary size. Such
patients usually give a history of repeated attacks of tonsillitis
in earlier years. They come to the physician for a more or
less constant pain in the throat which is aggravated by deglu-
tition, and it is frequently noted that the dysphagia is less
annoying when solids are ingested than when the attempt is
made to take liquids, an apparently anomalous condition which
attracts the attention of the patient. The patient may be aware
of a constant sensation as of a foreign body in the throat,
leading to repeated futile efforts to clear the throat with re-
sulting coughing or emesis. The breath is often fetid. The
voice may have a nasal quality.

Inspection of the tonsil region of such a patient shows a
unilateral swelling, if the calculus is large, with a variable de-
gree of inflammatory reaction. The first impression may be
one of neoplasia or of abscess. The swelling very frequently
centers about the upper pole of the tonsil in the region of the
supratonsillar fossa. Investigation with a probe reveals the
calcareous nature of the included mass, or the calculus itself
may be visible at the mouth of an enormously dilated crypt
or beneath an area of ulceration. If so, the central denuded
area will show a marginal velum of attenuated mucosa and
tonsillar tissue retaining the stone in position.

In nearly half of the reported cases the calculus has been dis-
lodged spontaneously or during the voluntary efforts of the
patient. Operative removal with the forceps, either with or
without incision of the retaining soft parts, has rarely been
difficult and as a rule is attended with surprisingly little opera-
tive trauma. A number of cases emphasize the tendency to
recurrence of such calculi in the same tonsil if tonsillectomy
is not done.

The accumulated literature establishes also the gross pathology of such calculi. They are usually rounded or oval, but with a somewhat roughened granular surface. More rarely they are mammillated or even coralliform. The possible range in size has been previously discussed. As to color, they are usually described as yellow or yellowish gray, sometimes light brown or greenish brown. A fetid odor has been almost constantly noted. Although very opaque to X-rays, a concentrically laminated or radially striated structure may be indistinctly shown. The consistency, while hard, is nevertheless usually qualified to the extent of calling attention to a moderate degree of friability such that small pieces may be broken from the main mass during removal. The specific gravity of tonsil calculi is given by Lange[36] as 1.715. The chemical analyses of tonsilloliths as given in the literature show them to be altogether comparable to similar calcareous deposits elsewhere. They consist essentially of alkaline, especially calcium, phosphates and carbonates with an organic matrix. Terrillon[59] quotes an earlier analysis by Langier as

Water	25.0	parts
Calcium phosphate	50.0	"
Calcium carbonate	12.5	"
Organic matter	12.5	"

100.0 parts

The organic matter is termed "mucus," with the added explanation that it includes epithelial cells and masses of leptothrix. The calculus described by Robertson[5] showed, on analysis:

Organic matter	18.4%
Inorganic	81.6%
Phosphoric anhydride	50.0%
Calcium and magnesium oxides	28.2%

Rosenberg[52] reports traces of iron, potassium, sodium and chlorin in addition to the major constituents. The close chemical relationship to concretions in general, and especially to salivary calculi, is evident. For instance, Harlay* reports the

*Harlay, M. V.: Calcul salivaire du canal de Wharton. Jour. de Pharm., et de Chim., 1903, xviii, 11.

following analysis of a salivary duct calculus, based upon the desiccated substance:

Organic matter 15.9%
Calcium phosphate 75.3%
Calcium carbonate 6.1%
Undetermined 2.7%

The incidence of the more minute calculi and the process of their development throughout the earlier stages and in association with various organic matrices have not hitherto been reported in detail.

Material and Method.—More than 16,000 pairs of faucial tonsils and about one-half as many pharyngeal tonsils have now been given routine microscopic examination in the pathologic laboratory of the University of Michigan. The methods used have been sufficiently described hitherto.* It need only be repeated that by means of a systematic search of relatively few large sections, each representing the entire cut surface of the longitudinally bisected tonsil, we have been able to give this material intensive study in spite of its enormous numbers. Upon a large part of this material the original pathologic diagnoses were made by Dr. A. S. Warthin, who has freely placed his accumulated data and experience at my disposal.

About three-fourths of the patients, from whom these tonsils were obtained, are included in the age group between five and twenty-five years, but the extremes of life are also represented. Except for the fact that there are included a large number of university students, many of whom have tonsillectomy done while in residence in Ann Arbor, the patients composing this group have not been selected by the operation for other than the usual factors which bring patients, either primarily or as refers from other departments, to the otolaryngologist for tonsillectomy.

Incidence.—As far as larger calculi, capable in themselves of producing clinical manifestations, are concerned, our experience sustains the general impression that they are compara-

*Weller, C. V.: The Incidence and Histopathology of Tuberculosis of the Tonsils. Arch. of Int. Med., 1921, xxvii, 631-660. The Incidence and Histopathology of Bone and Cartilage in the Tonsil. Ann. of Otol., Rhinol. and Laryngol., 1923, xxxii, 687-729.

tively rare. Only once during the period covered by this survey have we received a calculus which had been removed from a tonsil in the clinic. This calculus was small as compared to many described in the literature, and the case presented no unusual feature other than the presence of the calculus itself.

Calculi of somewhat smaller size, but not clinically recognizable, are frequently encountered in bisecting the tonsils for microscopic examination. These are macroscopic, ranging from such as are barely visible up to the size of a pea. The larger ones, therefore, approximate in size the smaller calculi which have been removed from the tonsil in situ, and there can be no doubt that we are dealing with a continuous series. As seen macroscopically these calculi of intermediate size are usually white or grayish white in color and frequently rather friable or chalky in consistency, so that they may be fragmented in cutting through the tonsil. It is evident to the naked eye that they lie in a dilated crypt or cystic space, from which they may be dislodged by the knife. No statistical record has been kept of the occurrence of calculi of this size, but we believe that an incidence of 2 per cent would be a conservative estimate.

Still more common are the calculi discoverable by microscopic examination alone. These are of such frequency that one need examine but a few tonsils in any series in order to find one or more examples. On the general principle that if search is being made for a particular structure its presence makes a more lasting impression than its absence, we feared that we might grossly overestimate the frequency of early calculus formation if an accurate tally were not made. Accordingly, a careful research of the sections from a series of 1,000 consecutive pairs of tonsils was made in order to have figures which were accurate in so far as they represent minimum values. (The actual incidence must of necessity be somewhat higher, since all portions of the tonsil are not examined.) Of these 1,000 cases, it was found that 80 showed calcareous deposits in the crypts of one or both tonsils, an incidence of 8 per cent. The average age of all the cases in the series of 1,000 was 18.7 years, while the average age of the 80 positive cases was 16.8 years. The difference between these figures cannot be considered as significant. The more frequent oc-

currence of larger calculi in adult life may be explained in part by the lapse of time required in their formation. Microscopic study does not indicate any special age of incidence in respect to the genesis of the smaller calculi. Likewise analysis of the series as to sex failed to show any difference in sex incidence beyond the range of error inherent in the limited size of the group of positive cases.

Pathogenesis.—Reference has already been made to some of the earlier views of the origin of tonsillar calculi. Terrillon[59] showed that they were not comparable to gouty deposits, a view dating back to Schenk (1609), in that they did not contain urates but were composed almost entirely of alkalin phosphates and carbonates. De Simoni[17] gives Robin (1874) credit for recognizing the part played by bacterial organisms in calculus formation, emphasizing branching organisms of the leptothrix group, and likens the part played by organisms in the formation of tonsil calculi to that of the tubercle bacillus in respect to pulmonary concretions. Less popular was the view of Gibb,[23] who did not hesitate to assign the origin of a tonsillolith the size of a pea to the resolution of a tuberculous deposit in the tonsil. Morroway[45] was inclined to associate the formation of the calculus reported by him with the use of very hard drinking water, the more so in that renal calculi, and also a parotid duct stone, had been removed from others in the same neighborhood. The repeated demonstration of an organic nucleus or, at least, of the admixture of organic material discoverable upon analysis, brought tonsillar calculi into closer agreement with other calculi in regard to mode of formation; and Compaired,[12] in 1904, stated that while the most admissible etiology is the predisposition created by the calculous diathesis, any foreign body, no matter how small, mucopus, bacteria, exudate, cyst contents, may determine lime salt deposit.

Calcareous deposits in the tonsil, as seen in microscopic sections, occur in various situations and in various organic matrices, as shown by the following table:

1. Within the tonsillar crypts, where they arise for the greater part in

    A. Colonies of mouth organisms.

    B. Keratohyalin.

C. Vegetable material (food débris).
D. Lipoids. (Cholesterin, etc.).
E. Inflammatory exudate, purulent or mucopurulent (pharyngeal tonsil).
F. Blood (hemorrhage).
G. Desquamated epithelium.

2. Within the lymphoid tissue, in
    A. Old abscesses.
    B. Old hemorrhagic areas.
    C. Caseous necrosis of tuberculosis.
    D. Langhans' giant cells of true tubercles.

3. Within the stroma and capsule.
    A. Chiefly in cartilage preparatory to endochondral ossification.

Among the group of 80 positive cases of microscopic tonsillar concretions of the crypts found in 1,000 consecutive pairs of tonsils, 47, or about 59 per cent, were developing in the large, socalled "actinomyces-like" colonies of mixed mouth organisms so commonly found in the crypts of the tonsil. This is the most common mode of origin. Miodowski,[43] as long ago as 1907, gave a good colored illustration of the early stage of this type. Such colonies grow peripherally with a more or less clearly rayed or clubbed outer zone. As they become of large size the central portion dies, and in the dead center lime salts may be laid down. At first, in the form of fine distinct granules, the mass becomes more dense as it becomes larger (Fig. 1), the diameter increasing only as more and more of the interior of the colony becomes necrotic (Fig. 2). As the concretion increases in size and becomes older, the finely granular character of the interior gives way to a coarse dense mass, producing somewhat angular fragments when it is shattered by the microtome knife in sectioning (Figs. 3 and 4). The peripheral portion of the colony may continue to live and grow, even after the concretion has reached considerable size. Such concretions in all stages of development give the usual reactions for lime salts. They are stained a deep blue to blue black by the hematoxylin stains, respond to the Von Kossa silver phosphate method and are dissolved by treating with mineral acids.

In thirty-three, or 41 per cent, of the 80 positive cases,
the origin of the tonsillar calculi was found to be in the accum-
ulated keratohyalin masses in the crypts. This mode of origin
is a very close second in point of frequency to the one just
described. When Hayton[28] presented his case of tonsillar cal-
culus in 1917, the presiding officer (Dr. A. Brown Kelly)
said that he doubted the occurrence of calculi developing
around leptothrix filaments, but that he believed that he had
evidence that they may develop around keratoses. These two
dissonant opinions, taken together, include the genesis of more
than 95 per cent of all tonsillar concretions, as found micro-
scopically. In the masses of keratohyalin so commonly found
in the crypts of the tonsil, calcification begins in certain of the
older hyalin laminæ so that such an area, when seen in micro-
scopic section, shows elongated striæ of lime salt, indicating
the roughly concentric layers of the original material (Fig.
5). Less often the interior of the keratohyalin plug may show
a granular deposit of lime salt, or through partial liquefaction
small lenticular masses of epithelial hyalin may be left and
these may subsequently undergo calcification. The striated
type of formation is exactly comparable to the calcification of
necrotic cells elsewhere in the body (kidney epithelium, heart
muscle, lung capillaries), in which the form is preserved by
the gradual impregnation and replacement of the original sub-
stance by mineral salts. In a later stage of the process, the
heavier deposit of lime salts completely effaces all evidence
of the original keratohyalin matrix in the central portion, while
the periphery may still show the concentric lamination char-
acterizing crypt contents of this type (Figs. 6 and 7). Such
calculi, while they are still small, may become very hard and
dense, so that they exhibit a coarse fragmentation when struck
by the microtome knife if they have not been previously de-
calcified.

The less common types of origin were not represented in
the test series, although they have been met with more fre-
quently in the total series than their nonappearance in 1,000
consecutive cases would seem to indicate. Vegetable material
(Fig. 8), coming from food remains which have been forced
into the crypt mouth and retained, may form the matrix of a
calculus. Before the lime salt infiltration is too heavy the

characteristic cellulose walls of vegetable cells at once indicate the nature of the foreign material. Robertson[31] believed this mode of origin highly improbable. It is frequently combined with development in colonies, for the vegetable material may be entirely covered by an extensive radiating colony of mouth organisms, as in the instance illustrated, although here there is no evidence that the lime salt deposit, which is occurring in coarse granules in the interior of vegetable cells, has in any way been determined by the organisms present on the outside.

With cystic dilatation of crypts, especially when crypt abscesses have developed, lime salt deposit may occur in various organic matrices, old pus, seropurulent, or mucopurulent exudate, desquamated epithelium or blood from hemorrhage into the crypts. Cholesterin is very commonly found in such crypt contents, and the lime salts are often laid down around or even replacing crystals of cholesterin (Figs. 9 and 10). Concretions of this type are found more frequently than any other in the pharyngeal tonsil. Inspissation of the crypt contents is not necessary for calculus formation. Granules or larger masses of lime salt may be present at the same time with hydropic change in the leucocytes or even marked liquefaction, or many of the leucocytes may show a fatty vacuolar degeneration.

The larger tonsillar concretions, subject to report from the clinical side, apparently have always had their origin in crypts. As in the lymph nodes and the spleen, however, calcification of masses of blood from hemorrhage or of the contents of encapsulated abscesses may occur in the lymphoid tissue of the tonsil. This is but rarely seen and lacks the clinical importance of the crypt calculi, which by their mode of origin offer the possibility of continuous growth by accretion. Calcification of areas of caseous necrosis of tuberculosis is very rare in the tonsil. No marked degree of this process, comparable to that so often found in lymph nodes, has been seen in over 400 examples of tonsillar tuberculosis diagnosed in this laboratory. The explanation must lie in the facts that extensive caseous necrosis is very rare in the tonsil and that, when present, it means a very virulent, rapidly progressing process, lacking the necessary chronicity. Calcification of the necrosing interior of Langhans' giant cells occurs more fre-

quently in tubercles in the tonsil than elsewhere in the body
(Figs. 11 and 12). The danger of erroneously interpreting
such formations as foreign body pseudotubercles has been pre-
viously pointed out (Weller[65]). The occurrence of three exam-
ples of this, in addition to those previously reported, has con-
firmed our earlier observations. Lime salts are laid down in
concentric laminæ in the necrobiotic interior of the giant cell.
Such concretions never reach a large size.

Within the supporting tissue and capsule of the tonsil the
only important occurrence of lime salt deposit is in connec-
tion with the development of bone and cartilage. Falling in
a quite different group, this process has been recently de-
scribed from this laboratory (loc. cit. p. 8) and need not be
further illustrated here. There is no connection between this
process and the formation of tonsilloliths in the clinical sense.

Concretions of microscopic size are frequent in the pharyn-
geal tonsil; large ones, rare. Here the most frequent mode
of origin is in the mucopurulent content of dilated crypts lined
by columnar epithelium (Fig. 9), but origin in colonies and
in keratohyalin masses is also often seen.

While in this discussion are included all of the important
sources of calcareous concretions in the tonsil, it must be borne
in mind that those concretions of sufficient size to be of clin-
ical importance are practically always primarily of crypt ori-
gin. There has been abundant clinical evidence of this in the
literature, and our own study of the origin of smaller calculi
capable of growing by accretion to a larger size confirms the
clinical observation.

It will be noted that in every variety of the formation of
calcareous concretions in the tonsil described above, with the
possible exception of the calcification of cartilage matrix, the
process begins in or upon necrotic organic material. Even
in the large colonies of mouth organisms in the tonsillar crypts,
it is only the dead interior which exhibits calcification. The pro-
cess therefore conforms in this respect to the general law of de-
velopment of calculi elsewhere in the body. Similarly, the gross
structural analogy is also carried out. Both by inspection of
the fractured surface and by X-ray examination there may
be shown the combination of concentric lamination and radial

striation characterizing most calculi, although this structure is never so clearly shown, as it is in many other calculi, especially those of biliary origin.

Microscopic study of tonsil calculi adds but little to the general chemical theories of calculus formation. With an organic nucleus provided and this nucleus in a state of necrobiosis or necrosis, the conditions are favorable for the deposit of lime salts. These can come only from body fluids in which they exist in a soluble form. Change in concentration, alteration in degree of alkalinity, escape of CO_2 upon surface exposure, change in the constitution of the phosphorus compounds and fixation of alkali through formation of soaps are all possibilities. Two of these, precipitation by loss of CO_2 and soap formation, seem most probable in view of the known conditions. That soap formation plays an important part is supported by the very frequent appearance of cholesterol in the microscopic stages of calculus formation (Figs. 5, 6, 9 and 10). It may well be that the fatty acid components of the esters from which the cholesterin was released have been fixed by soap formation, perhaps first as sodium and potassium soaps, substituted in turn by calcium and magnesium, and eventually losing their organic fraction to form the inorganic salts shown by analysis to be present in tonsil calculi.

The inflammatory reaction forming an important part of the case history of the larger tonsilloliths is found also around many of the smaller concretions. Purulent exudate frequently surrounds a small calculus, and the question may properly be raised as to whether the purulent inflammation or the calculus is the original condition. Where the calculus presses against the crypt mucosa ulceration may be shown, or there may be an inflammatory infiltration of the mucosa and submucosa of a degree much more marked than that present elsewhere in the same crypt. Whether the explanation be mechanical or bacteriologic, or both, there is microscopic evidence that the presence of even small calculi favors a local inflammatory process in the wall of the crypt and maintains such a reaction when once it is originated. Comparison might be drawn between this and the part played by fecal concretions in the etiology of appendicitis.

SUMMARY AND CONCLUSIONS.

1. While tonsilloliths large enough to attract attention clinically are relatively rare, smaller calculi are very common in the tonsil, showing an incidence exceeding 8 per cent of those submitting to tonsillectomy for all reasons.

2. Since all transitions in size exist between the smallest calcareous granules and tonsilloliths of large size, the genesis of the latter can be properly studied by microscopic methods.

3. Upon microscopic examination, 1,000 consecutive pairs of tonsils yielded 80 cases showing calculus formation in the crypts. This may be taken as the minimum incidence figure for the 16,000 pairs of tonsils which have now been examined in this laboratory.

4. Although clinical tonsillolithiasis occurs particularly in late adult life, no significant difference in age between positive and negative cases was shown by our series. The average age of the positive cases was 16.8 years. These facts considered together seem to indicate a relatively slow rate of growth for the larger calculi.

5. Microscopic study of the genesis of tonsillar calcareous concretions shows that such deposits may occur in any portion of the tonsil. With very rare exceptions, however, it is only in the crypts that concretions reach such a size that they become clinical tonsilloliths.

6. In the faucial tonsil the most common modes of origin within the crypts are in the dead interior of colonies of mouth organisms and in masses of keratohyalin. The former method is slightly more common than the latter. These are the chief sources of clinical tonsilloliths.

7. Vegetable material, food débris, inflammatory exudate, desquamated epithelium and old blood from hemorrhage may also serve as the organic nucleus for calculus formation in the crypts. With any of the substances mentioned, cholesterin may also be present.

8. In the pharyngeal tonsils, calculus formation occurs especially in mucopurulent exudate and in keratohyalin.

9. In the lymphoid tissue of the tonsil calcification of old blood, pus or caseous necrosis may occur, as in lymphoid tissue elsewhere. Small intracellular concretions in the giant cells

of true tubercles may cause them to be mistaken for foreign body pseudotubercles.

10. Tonsil calculi conform to the general laws of calculus formation in that they develop only upon an organic nucleus in a state of necrosis or necrobiosis, and that they may show, although but faintly, concentric lamination and radial striation.

11. The very common association of cholesterin crystals with the beginnings of calculus formation in the tonsil indicates that the presence of lipoids and the formation of soaps may be an important part in the chemical mode of their development.

12. As tonsillar calculi become larger, their presence favors the origin and continuation of an active inflammatory process in the wall of the crypt in which they are situated.

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Figure 1.   Photomicrograph of a colony of mouth organisms in a
faucial tonsil crypt.   Granular deposit of lime salts in the dead in-
terior of the colony.   Early stage of calculus formation.   Hemalum
and eosin staining.
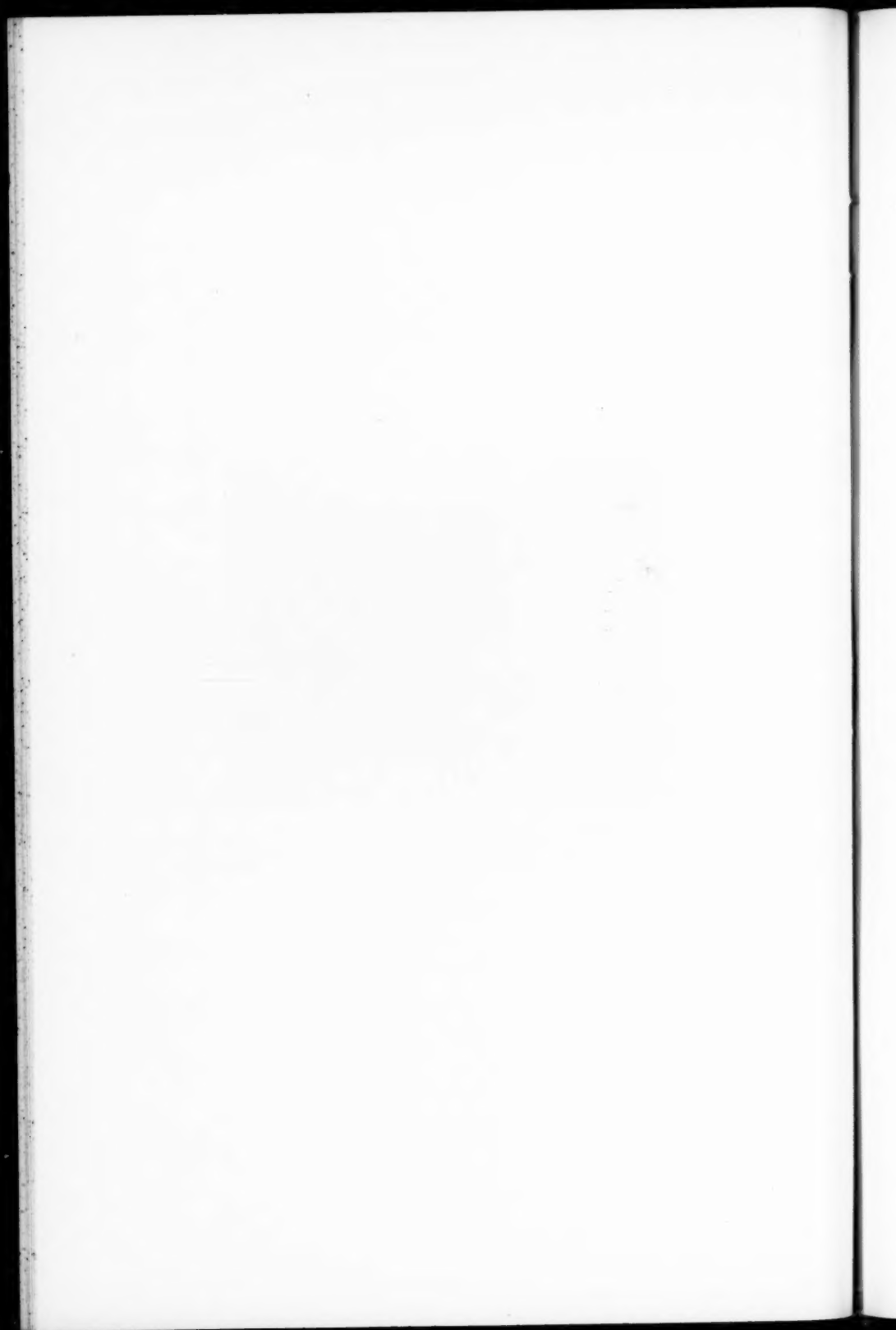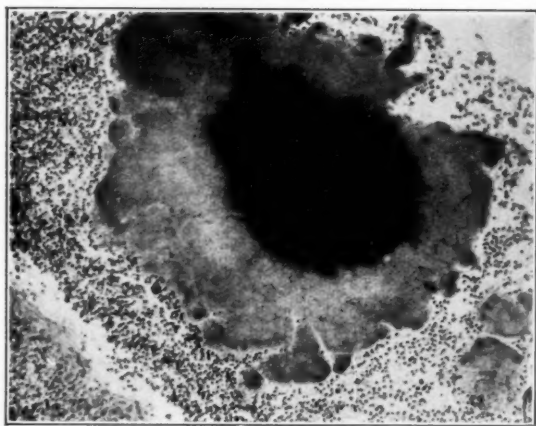
Figure 2. Older colony of mouth organisms in crypt of faucial tonsil. Extensive calcareous deposit in necrotic interior of colony. Purulent exudate filling the crypt around colony and concretion.
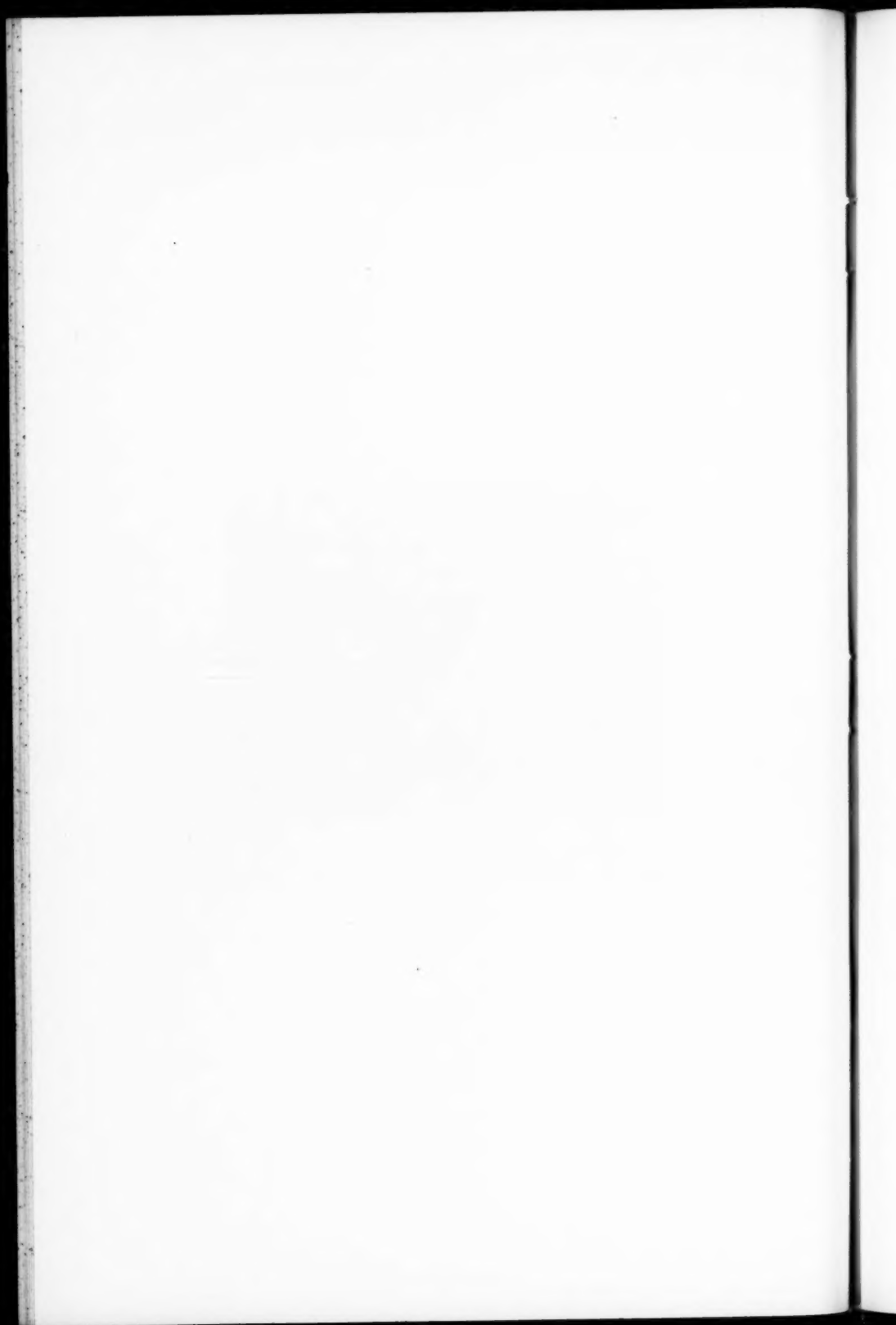
Figure 3. Photomicrograph of a somewhat later stage in the development of a tonsillar concretion in a colony in a crypt. The colony is still alive at the periphery. The concretion is more densely calcified than the preceding and has been coarsely fragmented and in part dislodged by the microtome knife.
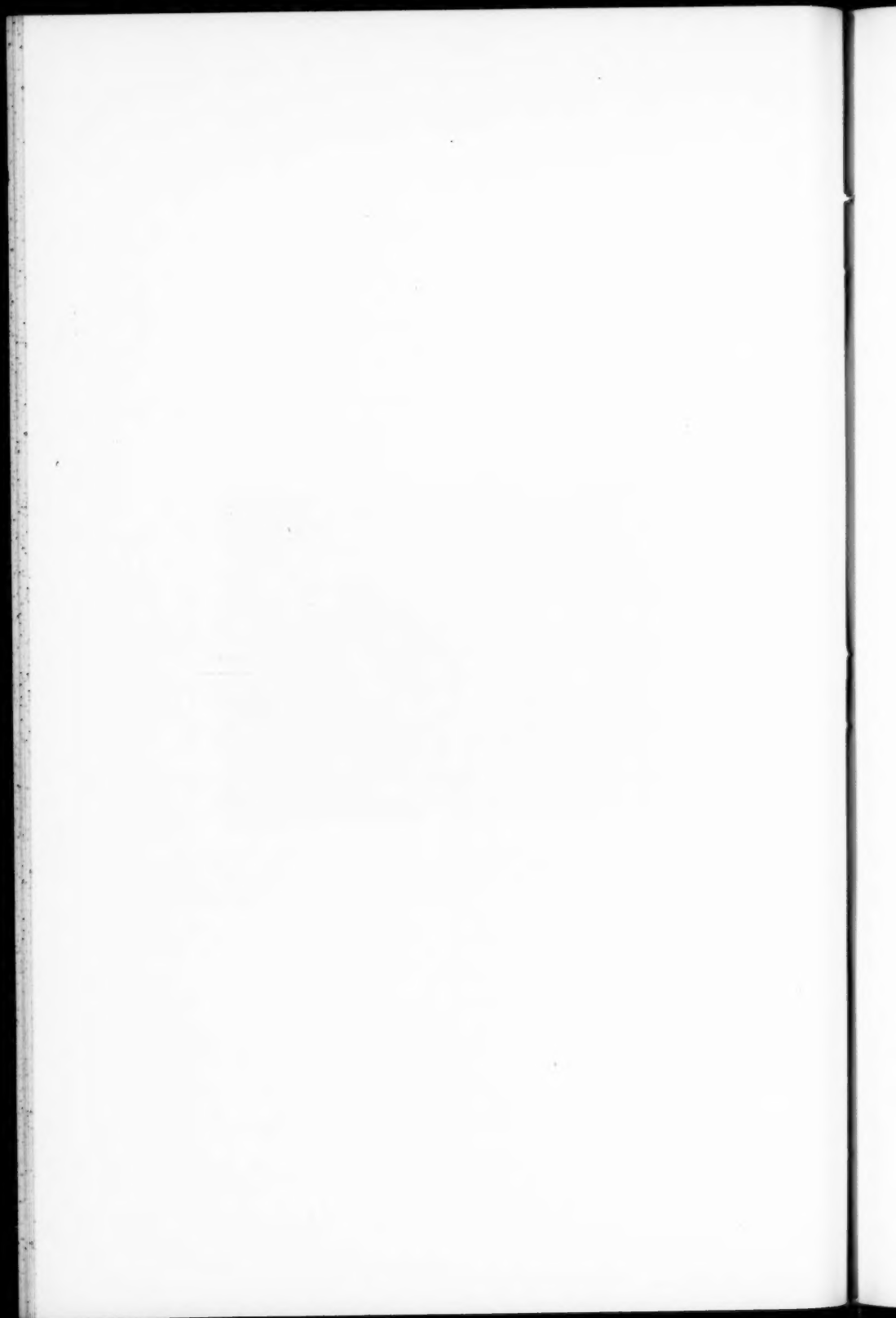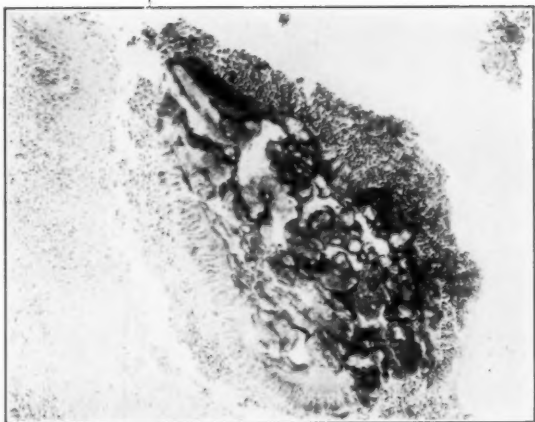
Figure 4. A later stage of a tonsillar concretion in a colony of which the border is still living. Coarse fragmentation during cutting. Purulent exudate over the surface.
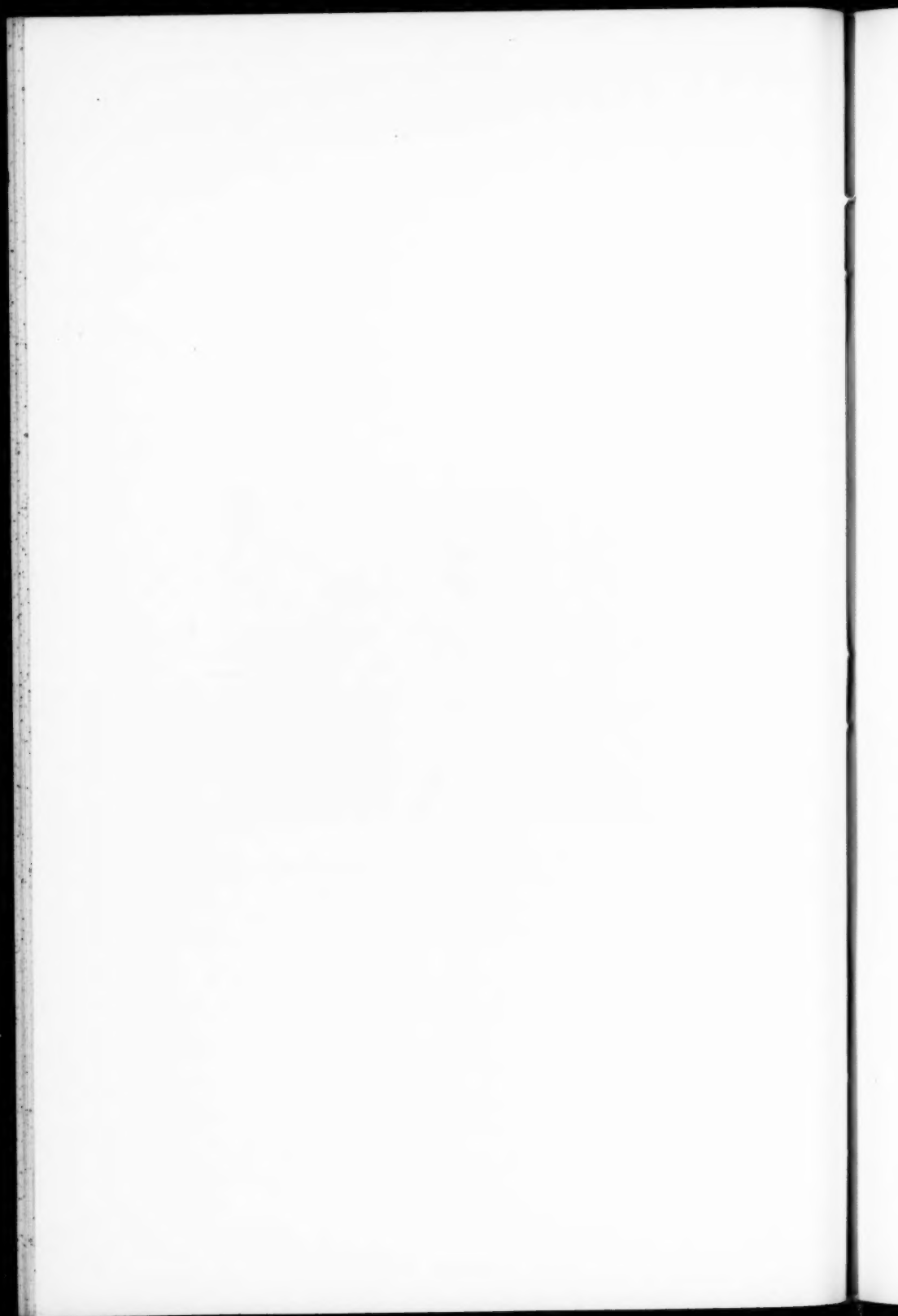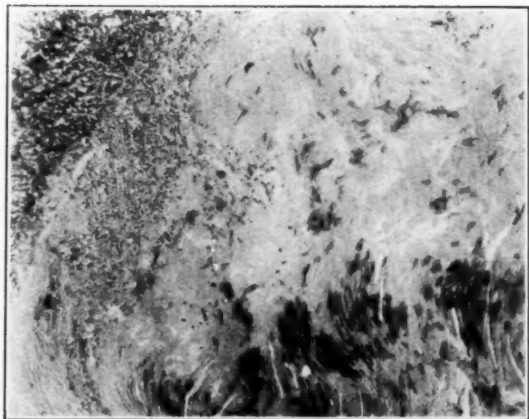
Figure 5. Photomicrograph of a very early stage of calculus formation in a mass of keratohyalin filling a crypt of the faucial tonsil. Mineral salts are laid down in certain laminae of the mass, without, as yet, becoming confluent. Note the clefts marking the location of cholesterin crystals throughout the mass.
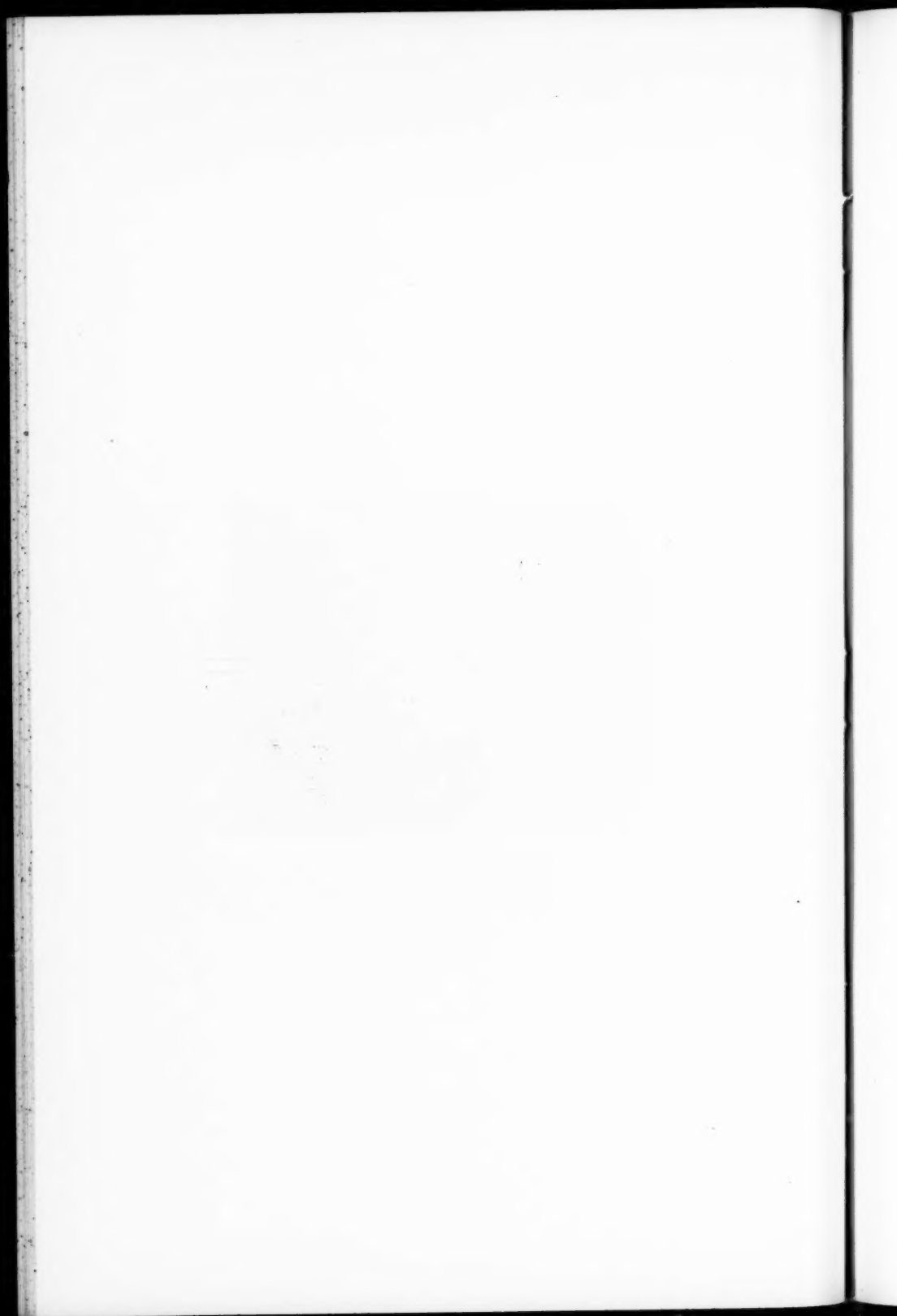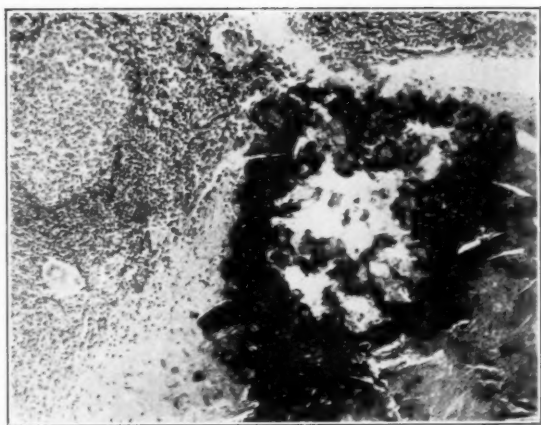
Figure 6. Later stage of calculus formation in keratohyalin in tonsillar crypt. The denser center of the concretion has been dislodged by the microtome knife for the greater part. The outer, and more recently formed, portion is still finely granular. This zone, and the surrounding keratohyalin, both show numerous cholesterin clefts.
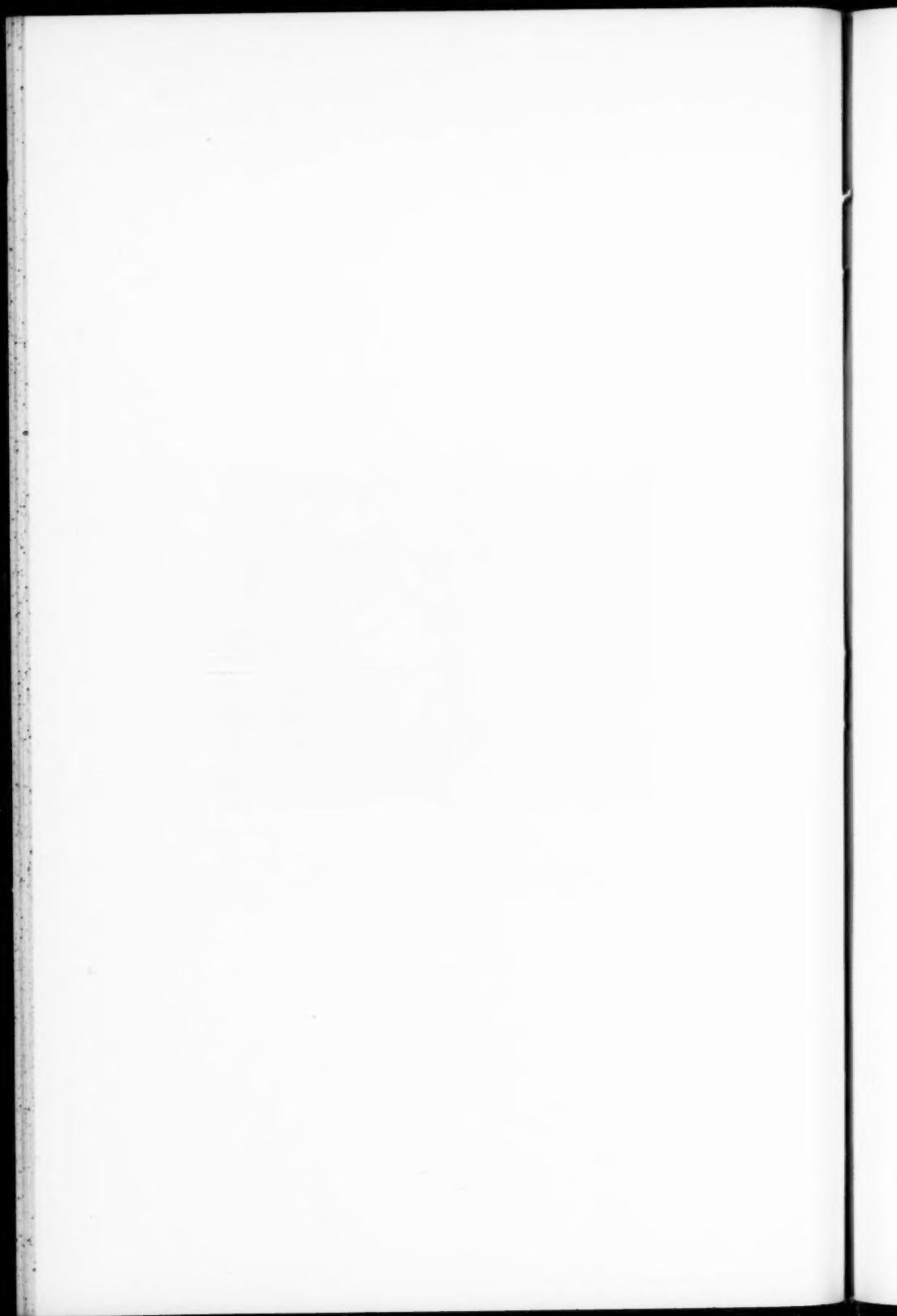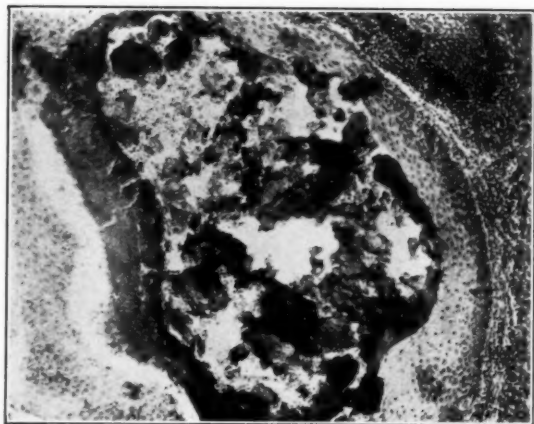
Figure 7.   Photomicrograph of a tonsillar calculus arising in keratohyalin and nearly filling the crypt.   Here the noncalcified border is narrow and likewise the more recent finely granular deposit.   The larger part was so densely calcified that it was coarsely fragmented when the tonsil was sectioned.
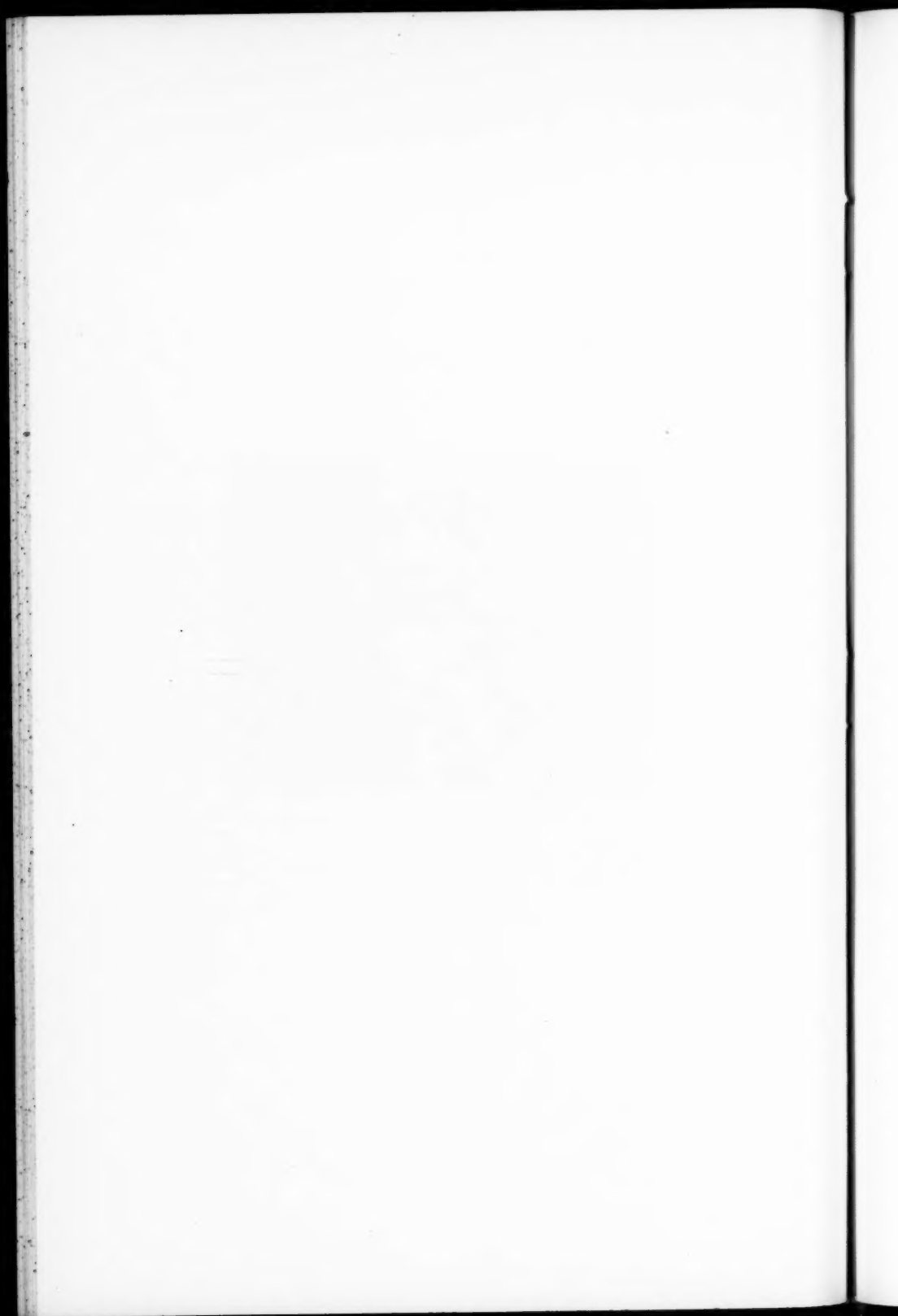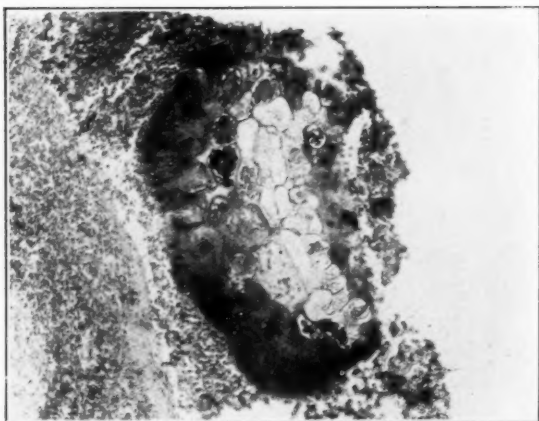
Figure 8. Vegetable food remains in a tonsillar crypt, covered with a living colony of mouth organisms, but showing early deposit of lime salts in the interior of some of the vegetable cells.
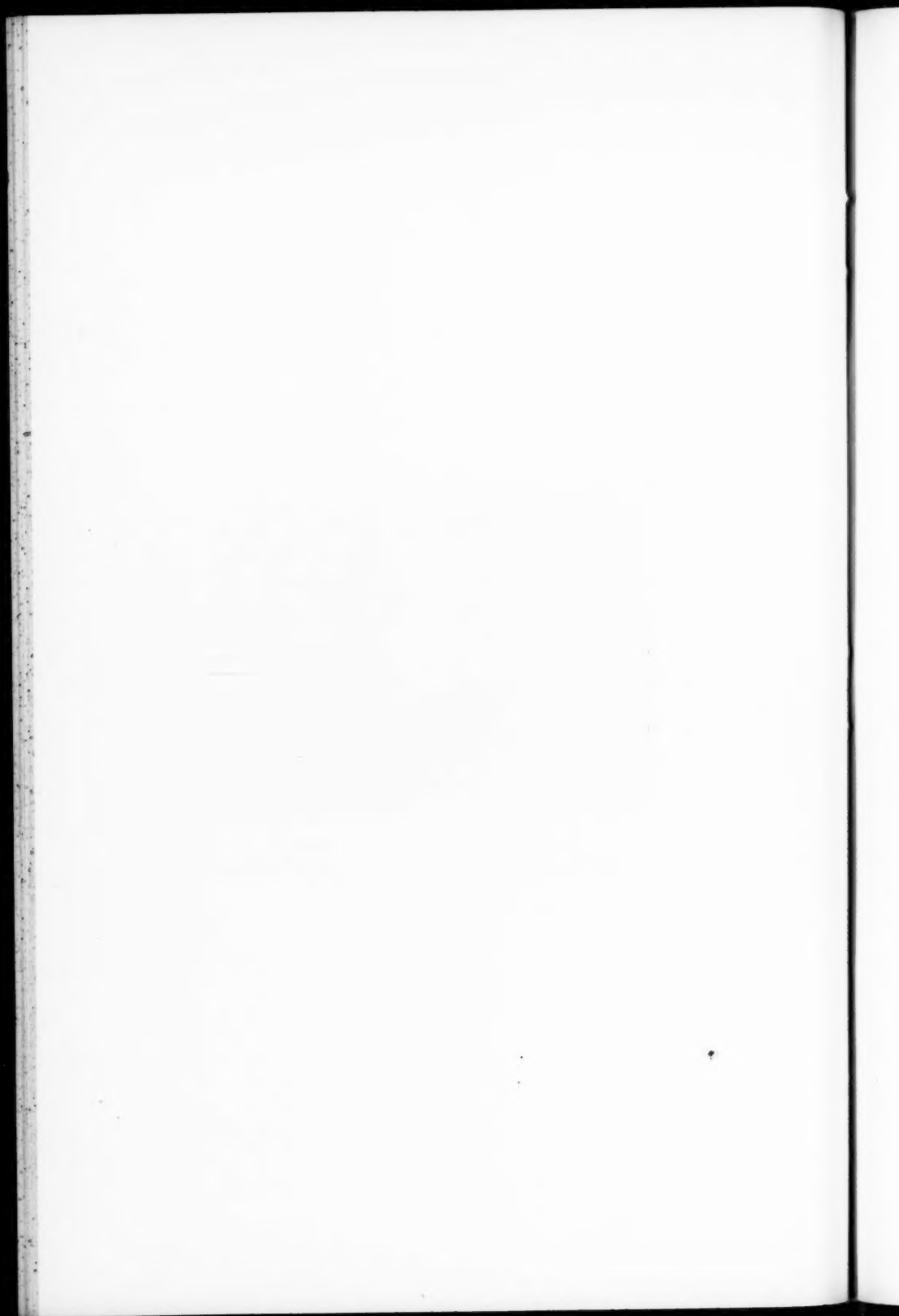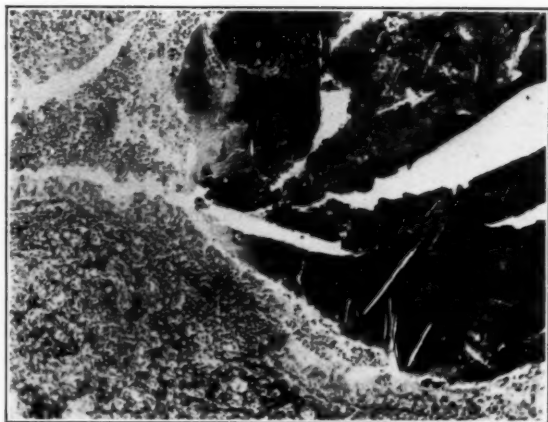
Figure 9. Large calcareous concretion developing in a crypt of the pharyngeal tonsil in a mass of mucopurulent exudate. Numerous cholesterin clefts. More recent purulent exudate about the concretion, and inflammatory infiltration of the mucosa.
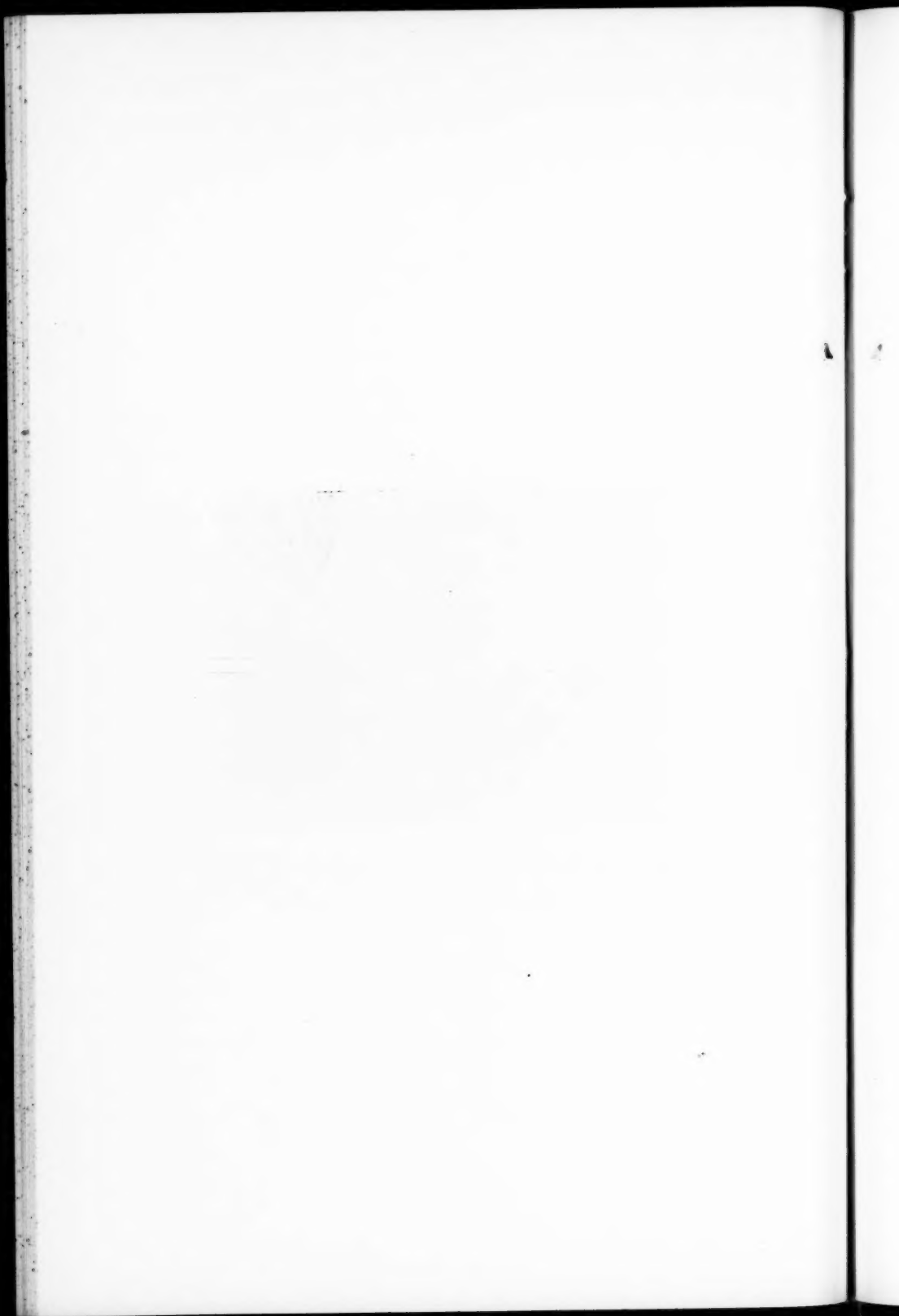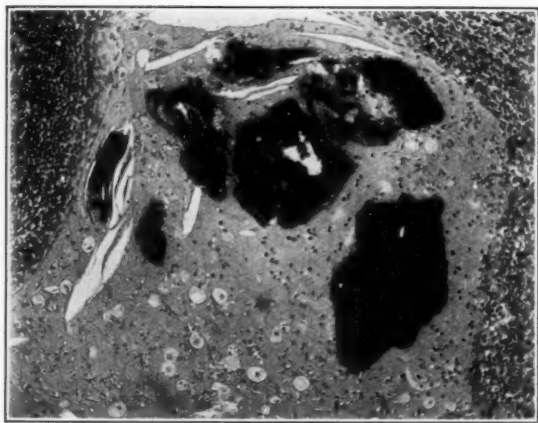
Figure 10.   Photomicrograph of liquefying purulent exudate and desquamated epithelium in a crypt of the faucial tonsil.   Calcareous concretion formation is occurring in association with cholesterin. Some crystals have been entirely covered with, or replaced by, lime salts, while others show an early stage of this process.   Fatty change of leucocytes.
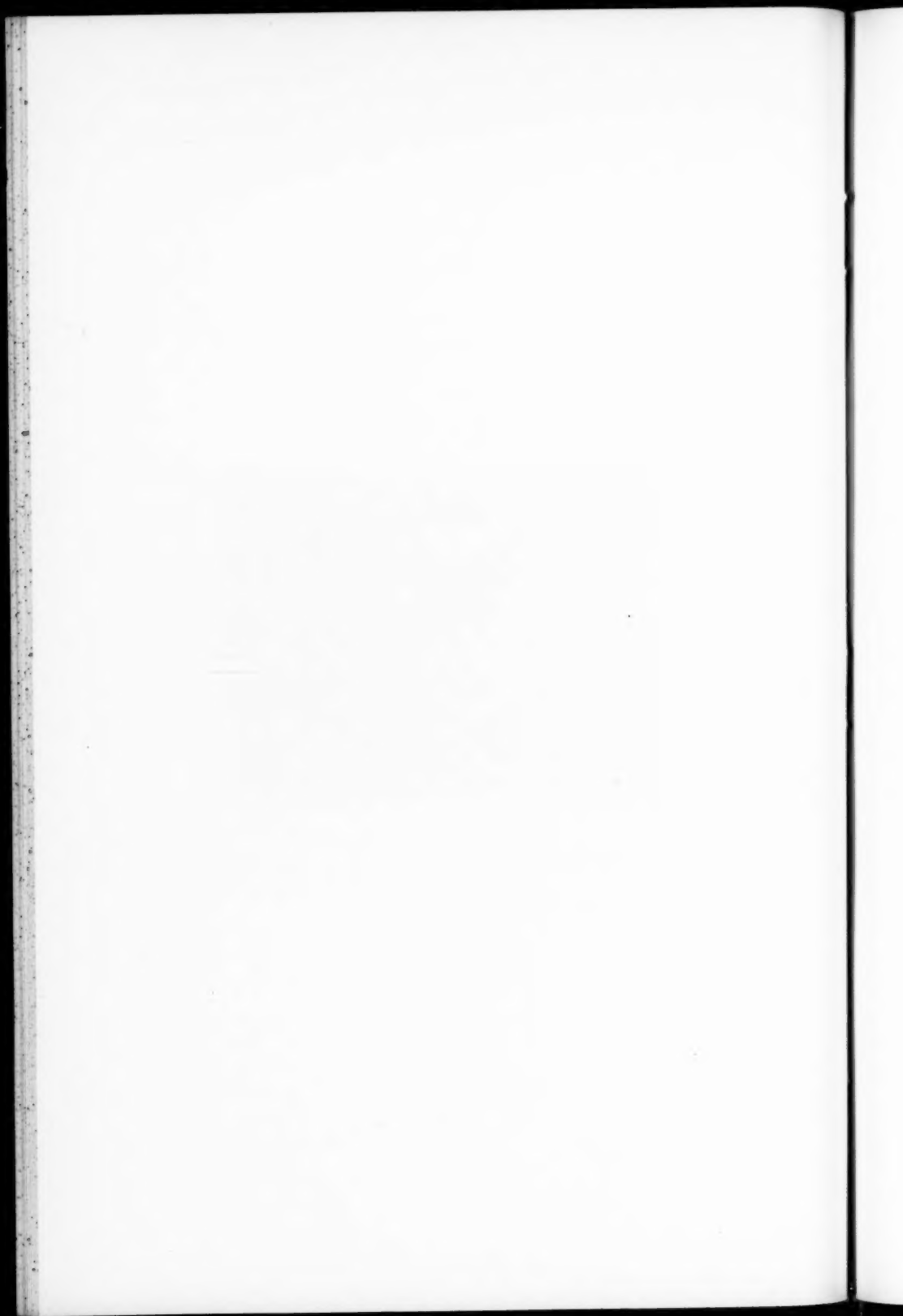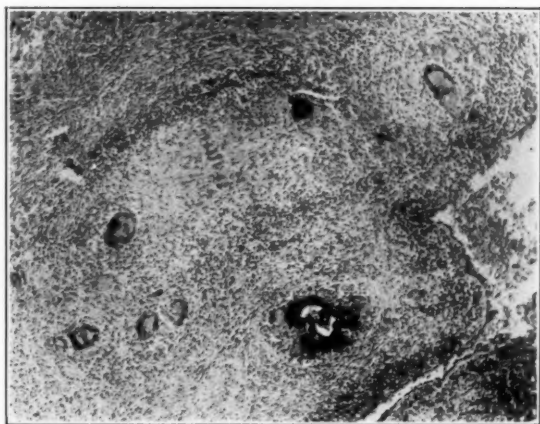
Figure 11.   Photomicrograph showing concentrically laminated
concretions in the interior of giant cells in tonsillar tuberculosis.
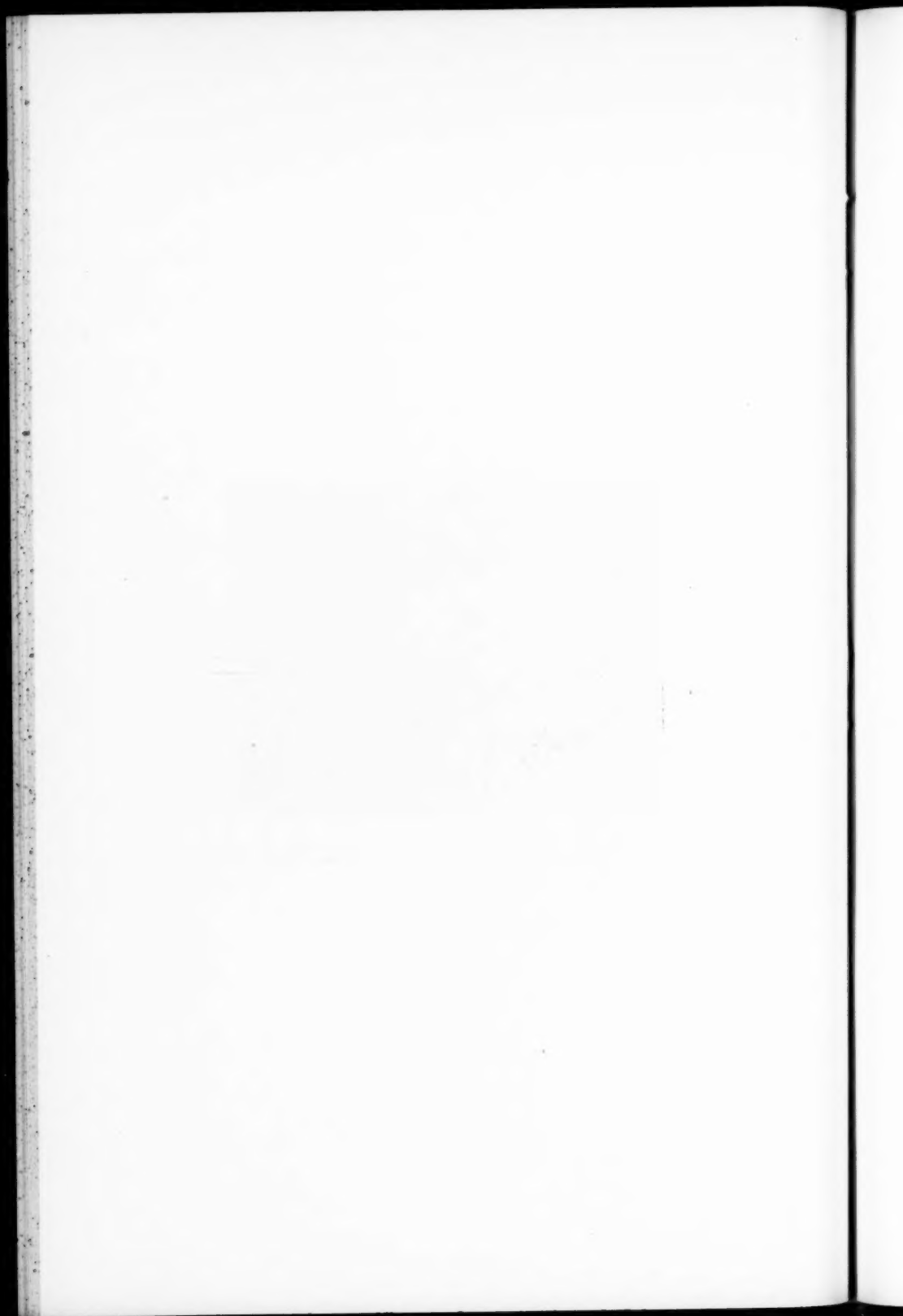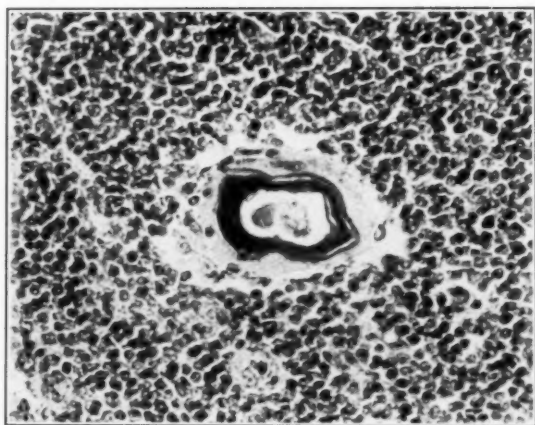
Figure 12. Photomicrograph at a higher magnification of laminated concretion in a giant cell of tonsillar tuberculosis. A central organic nucleus still persists. Outside of the calcareous laminae there is a zone of cytoplasm not yet calcified, but showing coagulation necrosis.
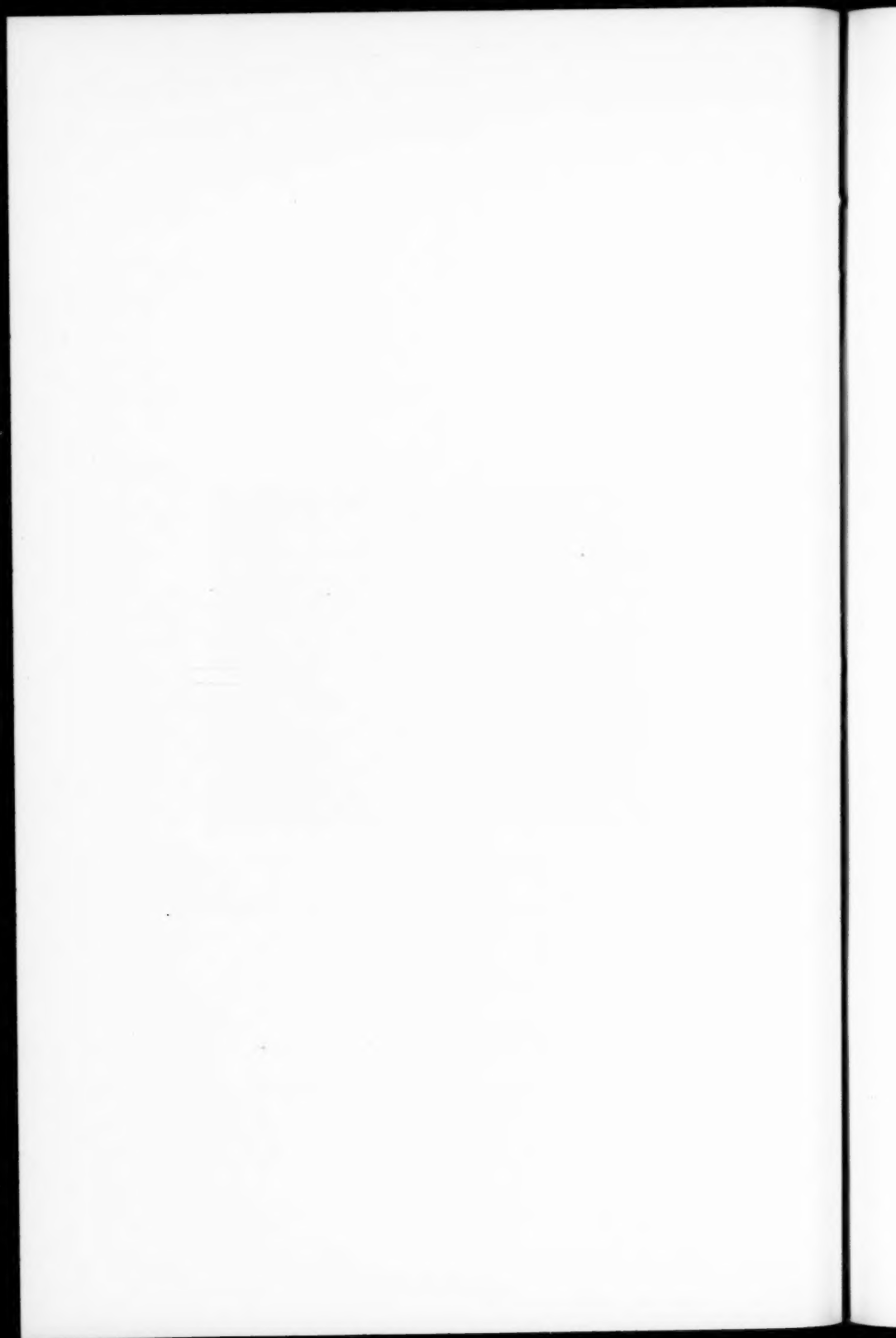
III.

AN ANATOMIC AND X-RAY STUDY OF THE OPTIC CANAL IN CASES OF OPTIC NERVE INVOLVEMENT.

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One is often asked why one person out of many having infections should lose his vision while others escape, and why one person should remain permanently blind and another recover spontaneously. This paper is an attempt to answer these queries.

That the osseous canals normally afford great protection to their neural contents cannot be doubted, but when these canals become narrowed and the enveloping sheath of the nerve, or the nerve itself, swollen, the unyielding walls may cause constriction sufficient to produce atrophy. While this fact has been generally recognized, the size of the canal has received scant attention.

Piersol gives the diameter of the optic nerve as from 3 to 4 mm. In a canal of 4 mm., therefore, only a moderate swelling would cause a pressure so severe that degeneration of the nerve would rapidly ensue.

The importance of filming the optic canal to determine adjacent pathology has been emphasized by van der Hoeve. This study was undertaken partially to substantiate his assertions but more especially to determine if there was any relationship between the size of the canal and the vulnerability of its contents. Many skulls were examined at the Harvard Medical School, thanks to Doctor Harris P. Mosher and the anatomic department. The results were tabulated and the size, shape and position relative to pneumatization were noted. Roentgenograms were made of the optic canals in all available cases of optic nerve involvement, old and new, to determine the size and shape in the old cases, and in the new ones to also ascertain any evidence of pathology. As a check radiograms were made of twenty-five supposedly normal individuals. For

this X-ray work I am indebted to Doctor A. S. Macmillan. At
the end of this paper I report fourteen cases numbered from
35 to 49, to correspond with my previous reports.

Van der Hoeve, after prefacing his remarks with the apt
statement that "the diagnosis of an existing sinus disease,
especially of a posterior sinus disease, is not always easy—
we may say, not always possible, and that the absence of any
sign of disease inside the nose is not sufficient to exclude sinu-
sitis from the diagnosis," goes on to say that "we happily have
support in the examination with the roentgen rays" and lays
special emphasis on the value of images of the foramen opti-
cum. "If I find a normal foramen," he continues, "I do not
believe the canal can be much deformed, but care must be
taken not to misinterpret a picture the result of wrong posi-
tion." He claims that the great advantage of the method lies
in the examination of the ethmoid in posterior ethmoiditis
adjacent to the canal, and cites a case of retrobulbar neuritis
and choroiditis which, although operated upon for sinus dis-
ease several times, was not cured until diseased posterior
ethmoid cells were opened, which were discovered in a radio-
gram of the optic canal. "Thus we see," he says, "that
roentgen photography may be of great support in the diagnosis
of sinus disease, and yet there are cases (he wisely adds)
which are not discovered even with roentgenography." In
concluding this phase of his paper, van der Hoeve well says:

"All cases must be submitted to X-ray examination by a
radiologist experienced in the skiagraphy of the nasal sinuses.
But even X-ray examination is not infallible, so that in the
presence of serious orbitoocular trouble, such as optic neuritis,
for example, the only satisfactory method of excluding sup-
puration of the sphenoid and posterior ethmoid cells, the sin-
uses most likely to set up eye disturbances, is to open them."

Up to the present I have been able to find but little evidence
of pathology adjacent to the optic canal in the X-ray plates.
Probably this is due to the types of infections responsible for
the neuritis in the cases observed since this study was com-
menced, as these happened to be largely from tonsil infection.
I regret, moreover, that I cannot quite agree with van der
Hoeve as to the importance of finding purulent sinusitis adja-
cent to the canal. It has been rarely possible, in my experi-

ence, to discover acute infections within the posterior sinus by
the X-ray, while in the chronic cases which show pathology
in the film, nature has walled off the infection from the nerve.

The important thing to determine in a study of the optic
canal is its size and shape, as this is always possible. Adja-
cent pathology, while always sought, is not so easy to deter-
mine, and many cases undoubtedly require surgical interfer-
ence where it is not evident.

The average diameter of the optic canal is given by Whit-
nall as from 5 to 6 mm., while Wilbrand and Saenger gave the
average as high as 7 mm.

The canals were measured in 164 skulls: 20 of these canals
were 6 mm. in diameter; 90, 5.5 mm.; 164, 5 mm.; 20, 4.5
mm.; 3, 4 mm; the average being 5.17 mm. The 5.5 and 6 mm.
canals were all round, and only two had extensive pneumatiza-
tion about them. Seven of the 164 canals of 5 mm. were slight-
ly oval. Four were triangular and but twelve showed extensive
pneumatization.

Of the 4.5 mm. canals (twenty in number), twelve were
oval or flattened at the top, and practically all were from two-
thirds to three-quarters surrounded by pneumatic sinuses.

The three canals of 4 mm. were all oval or irregular. Two
were nearly surrounded by pneumatized sinuses. The third
was in a very dense skull and was probable due to hyperostosis.

The canals were usually found to be round, irrespective of
the way examined—i. e., by holding them up to the light, by
the fluoroscope or the X-ray. The top posteriorly was occa-
sionally flattened, and in a few cases the canal appeared dis-
tinctly oval, with the horizontal diameter always the larger.
These findings differ but slightly from the description given
by Whitnall: "The anterior opening of the canal is oval in
outline, the central part circular, and the posterior opening
generally considerably flattened from above downwards."

I only occasionally found the posterior opening flattened.
At the end of Whitnall's description of the contents of the
canal he mentions that "the artery is embedded in the dural
sheath on the lower and lateral side of the nerve, and ossi-
fication of the fibrous septum between them accounts for the
rare abnormality of a doubled optic foramen." Three of
these double optic canals were found.

The hollowing out of the roof of the canal by an extension
of the pneumatization of the sphenoid or posterior ethmoid
over the canal was found eight times. Pneumatization of the
bridge of bone formed by the lateral root of the lesser wing
of the sphenoid which separates the optic canal from the
superior orbital fissure was noted four times. This is usually
found where the canal appears triangular. No optic canal was
discovered entirely surrounded by the sphenoid sinus, although
there is such a specimen in the Oxford collection. Several,
however, were nearly surrounded. Early in the measurements
it became apparent that excessive pneumatization of the sphe-
noid adjacent to the canal was usually associated with diminu-
tion in its caliber. It seems that extensive pneumatization
encroaches upon the canal. I found that Schaeffer had re-
marked as follows on the point: "If the root of the lesser
wing of the sphenoid bone is partially hollowed out, the optic
foramen and nerve are encroached upon."

The vulnerability of the contents in the optic canal, practi-
cally surrounded as they frequently were by pneumatic sinuses,
many times led me to ask why are not these troubles more
frequent? That others have been equally impressed, let me
quote Skillern:

"When reabsorption occurs in this direction the sinus en-
croaches upon the optic nerve, often to such an extent that the
nerve comes to lie almost within the sinus cavity. The im-
portance of this anatomic configuration cannot be overestimat-
ed, especially in connection with ophthalmic complications
resulting from infection of the nerve through inflammation of
the sinus mucosa."

Schaeffer along this line also says: "When the lesser wing
and the anterior clinoid process are partially hollowed out, the
sphenoid sinus encroaches upon the optic nerve, and if the
pneumatization is extensive the nerve lies, in a sense, within
the cavity of the sphenoid sinus. The clinical importance of
this relationship cannot be overestimated."

The thinness of the bone separating the inner wall of the
optic canal from the sphenoid was noticeable in many speci-
mens, and in a few dehiscences were found, but as these might
have resulted from cleaning the skulls it was thought best not
to tabulate them. Suffice to again quote Schaeffer: "In many

cases the nerve and vessels are separated from the mucous
membrane of the sinus by the merest shell of bone. Indeed,
osseous dehiscences may be present."

The size of the optic canals in infants and children is most
instructive. The skulls of forty stillborn infants were exam-
ined fluoroscopically. Of these it was possible to make out the
canal in twenty. In two, the canal was oval, and in the rest
approximately round. In none was it less than 4½ mm. in
diameter.

Thus it would appear that the diameter of the optic canal at
birth is nearly of adult size but very short. There is a rather
close analogy between it and the annulus tympanicus. The
canal seems to reach adult size early in life, judging by meas-
urements obtained from a few children's skulls. Of six canals
in skulls from children under three years, four were round
and two slightly oval and all approximately 5 mm. in diameter.
The four canals from skulls of children seven to eight years
old were all round and all of about 5 mm. in diameter. The
canals did not appear in these few specimens to be in very
intimate relationship to the pneumatic sinuses. Uniformity in
the radiograms was obtained by having all the work done by
one radiologist, Dr. Macmillan.

Of the fifty radiograms of the optic canal in the normal
series, five were slightly oval—i. e., the vertical diameter was
½ mm. shorter than the horizontal. Ten appeared oval, due
to faulty position. The average diameter of all fifty canals,
irrespective of faulty position, was 5.35 mm.

In the twenty-five cases with optic nerve involvement there
were twenty-six oval canals of which only two were considered
as due to faulty position. This makes practically 50 per cent
oval as against 10 per cent in the normal series. Several of
these were filmed a second or third time in an endeavor to
get correct positions. In ten there was 1 mm. difference be-
tween the vertical and horizontal diameters. In the others,
about ½ mm. In nineteen of these twenty-five the optic nerve
was involved. Of the twenty-four remaining canals (i. e.,
round ones) there was involvement of the nerve in twelve.
In the nineteen oval canals with optic nerve involvement, five
were bilateral. Twelve were operated upon, one recovered
without operation, and in the other where there was no oper-

ation there was no improvement. Of the ten patients with round canals one recovered without operation, six had intranasal operations, and in three the tonsils were removed.

The measurement of the optic canal on the X-ray films in ten cases (4, 6, 13, 23, 26, 27, 28, 33, 36, 42) showed a vertical diameter of 4 mm. or less. Two of these (4 and 13) were not benefited, or but slightly by operation. One had complete optic atrophy and remained totally blind. This case, by the way, had the smallest canals measured (3.5 by 4 mm.). The fourth refused operation and remained unimproved. Four in whom considerable improvement followed the operation, had marked pallor of the nerve head. The ninth case required a secondary operation, and the tenth one, unoperated (because of very poor general health), has had frequent recurrences.

Ten cases (4, 7, 8, 9, 24, 25, 26, 34, 41, 47) had optic canals which in the X-ray films showed a vertical diameter of 4.5 mm. and all but two made excellent recoveries and these two improved somewhat.

In six cases (2, 31, 38, 44, 46, 49) the X-ray films showed canals of 5 mm. or more vertical diameter, and in all practically normal vision was obtained.

The average diameter of the canals of the patients with optic nerve involvement was 4.45 by 4.92 mm., as contrasted with 5.18 by 5.52 mm. in the normal series. In six cases the right canal was larger than the left, and in four the left was the larger. In every unilateral case the smaller canal was the one involved. When both eyes were affected it was on the worst side. This seems to demonstrate that the smaller the canals the more serious the neuritis.

Schuller notes that "the occurrence of periodic paralyses and periodic neuralgia of the cranial nerves may perhaps be explained occasionally by the abnormal position or size of the nerve canals."

While but one case of hyperostosis was encountered it should not be overlooked that such a condition might cause loss of vision.

From Onodi, the source of so much valuable data on optic nerve disturbances, the following is quoted:

"Through postmortem findings, Virchow and Manz were able to trace the cause of blindness back to arrested develop-

ment of the skull in earliest youth, to a premature ossification
of the skull, to a hyperostosis, and in consequence thereof an
irregular contraction of the foramen opticum and pressure on
the optic nerve."

As the optic canal is located between the roots of the lesser
wings of the sphenoid, any change in the normal plane of the
wings would naturally produce changes in the position and
direction of the canal, and while no skulls were found or plates
examined which showed this abnormality it is nevertheless a
possibility.

Schuller says: "The lesser wings of the sphenoid, which
normally run lateral from the median line outward in the plane
of the planum sphenoidale (that is, in a horizontal direction).
show a steep rise directed upward laterally. Corresponding
to that, the position and direction of the canalis opticus and
the fissura orbitalis superior are in most cases almost abnor-
mal. Due to the fact that the course of the lesser sphenoid
wing is also directed upward, a kinking or a narrowing of the
lumen of the canalis opticus may result, since the latter lies
between the two parts of the former."

There is one other condition—scaphocephaly—wherein optic
nerve disturbances may depend on the peculiar shape of the
head and where roentgenograms greatly aid in diagnosis, as
they often show signs of craniostenosis.

The following is Dr. Macmillan's description of the correct
position for filming the optic canal:

The position in which a patient is placed for a radiograph
of the optic canal is of extreme importance, as a slight varia-
tion in the position would so change the outline of the canal
as to render its measurements worthless. Holding a dry
skull in the hand and looking at the optic canal through the
orbital cavity, it is evident that the true shape and size can
be seen only when the axis of the optic canal goes through
the lower outer quadrant of the bony rim of the orbit. The
optic canal in the living can be thrown into this area when
the patient is lying face down, with the malar bone, nose and
lower jaw touching the plate, the central ray directed straight
down. If the upper rim of the orbit is allowed to touch the
plate, the optic canal will be thrown too high in the bony frame
of the orbit and will be distorted. The diameter of an optic

canal of 5 mm. would be enlarged about ½ mm. in the radio-
graph of a living subject.

Case 35.—F. L. E., 27, referred by Dr. Peter H. Thompson
on October 8, 1921, with diagnosis of postneuritic or second-
ary optic atrophy. Good general health, no colds, tonsils re-
moved two years ago. Occipital headaches for some years.
Four months ago pain commenced about right eye. Felt as
though pressure from behind was pushing it out. Vision,
20/200 right, 20/70 left. Sees double. Visual fields show
contraction both for form and color. Both discs blurred, more
marked on right. X-ray report of accessory sinuses was nega-
tive; some infection in teeth. Septum deflected to right rather
far back, crowding right middle turbinate. Operated upon
October 19, 1921. Right middle turbinate removed; ethmoid
cells found projecting into nasal cavity. These were broken
down quite extensively. From the posterior one a large mass
of soft tissue (probably a cyst) was removed. Good sized
opening in front wall of sphenoid. Patient progressed favora-
bly. Pains in occipital region relieved and some improvement
in vision. April, 1923, vision, right eye 20/60, left 20/30.
Dr. Verhoeff reported on the turbinate tissue that the upper
part was polypoid and moderately infiltrated with chronic in-
flammatory cells.

Case 36.—R. R., 27, referred by Dr. H. B. C. Riemer on
October 19, 1921, with diagnosis of optic neuritis left. Rather
poor health, many colds and hay fever; no headaches. Two
weeks ago noticed soreness in left eye on movement, which
continued to get worse. A week ago blurriness when looking
straight ahead, but lateral vision good. The blurriness in-
creased until two days ago, when vision was finger movements
and light perception. Physical, neurologic and Wassermann
all negative. Septum deflected to right (opposite side from
eye trouble), with compensatory hypertrophy of the left mid-
dle turbinate. Turbinate very large and obstructive, at places
wedged tightly between septum and ethmoid wall. Films of
teeth and paranasal sinuses negative. Disc red and swollen
with blurring of edges and tortuosity of veins. Three or four
inferior molar roots were extracted, but no apical involvenent
was apparent. Left middle turbinate on removal was found
very large. Posterior portion of nose was extremely narrow,

and a considerable area of the posterior ethmoids was removed before the sphenoid could be reached. The lining of the sphenoid did not appear especially changed. The sphenoid septum extended far over to the right. The posterior ethmoid was very large and the wall seemed considerably thickened. Pathologic report by Dr. Verhoeff showed slight hyperplasia of connective tissue of sphenoid and slight infiltration with eosinophiles. Marked edema of submucous tissue of ethmoid and marked infiltration of latter with eosinophiles and slight perivascular infiltration with lymphoid and plasma cells. Three days after operation the patient could count fingers at six inches. In two weeks the disc was less blurred, outlines well defined, color smoky gray, temporal half slightly paler than normal, retinal vessels normal. Vision gradually improved and a month after operation was 20/80; a month later 20/40, and on January 1, 1923, was 20/30. The left optic canal was 4 by 4.5, the right 5 by 5.

Case 37.—G. L. H., 32, referred by Dr. George H. Ryder on March 9, 1922, with diagnosis of retrobulbar neuritis both. Very poor general health, just recovering from several serious pelvic operations. Six weeks ago first complained of eyes being lame and sensitive. Was unable to see things clearly; vision, fingers 2 feet right, 1 foot left. At present they are sensitive to strong light. No headaches, but tired and dizzy. Can distinguish with difficulty people across the room. Fundus practically normal, possibly slight temporal pallor. Septum was deflected rather far back, somewhat crowding right middle turbinate. Condition of eyes was thought, however, to be due to infection from pelvis and the prostration following operations, so nothing but mild nasal treatment was advised. Neurologic examination by Dr. Ayer, while practically negative, mentioned the possibility of a brain tumor.

Comment.—This case is reported to emphasize the importance of not operating on patients of this type. The woman's mental condition was very much upset. The physical condition was at the lowest ebb, and anything in a surgical way would have been a grave error. She was later seen by Dr. Verhoeff, who considered her trouble largely hysteria. Vision has gradually improved, and on September 10, 1922, was 18/200 right and 18/100 + left.

Case 38.—G. N., 19, referred from the eye clinic by Dr. Hatch on March 22, 1922, with diagnosis of choroiditis with optic neuritis left. Good health except persistent head colds. When awaking two weeks ago found a black cloud to left and above the line of vision in left eye. When first examined by Dr. Hatch vision was normal. Since then it has been failing so that yesterday it was 20/200 and this morning only the top letter on the chart. Large central scotoma. No pain or discomfort in moving eye. Several vitreous opacities. Wassermann negative, as was also the physical and neurologic examinations. Septum deflected to the left high up and far back. Left middle turbinate somewhat enlarged and in contact with septum. Moderate obstruction to posterior sinuses. Under treatment with argyrol and adrenalin spray there was rapid improvement, and in one week vision was 20/60 plus 2; in another week, 20/40; a month later, 20/20 plus, vitreous clear, slight blurring of edges of disc remained; six months later vision was normal, but fundus still showed a very slight haze. Both optic canals were 5 by 5 mm.

Case 39.—L. G., 18, referred by Dr. Horrax, Brigham Hospital, with diagnosis of retrobulbar neuritis right with question of brain tumor. Poor general health, no serious illness. Has had profuse nasal discharge for ten years. Complains of inability to walk well—"feet don't seem to go where I put them." Four months ago noticed sight in left eye was very poor. Had to use right eye for reading. This continued for three months. Then right eye began to fail and for past month has been worse than left. During past week her gait has been unsteady and she bumps into things. Considerable nausea for two weeks, also very severe headache. Was treated at Brigham Hospital. Examination of eyes there was "pupils fairly well dilated; react promptly to light and accommodation; moderate suboccipital tenderness left; persistent nystagmus, coarse to left; ataxia of hands and outward pointing error left; positive Romberg, staggering gait, hyperesthesia, left face; discs outline hazy and optic cups filled with edema; left disc rather pale; veins greatly engorged and somewhat tortuous." X-rays of Brigham Hospital were interpreted as follows: "Stereoscopic films of skull in left lateral position showed skull to be symmetrical; sella turcica itself is normal and there is no

definite evidence of increased intracranial pressure; skull very thin; sinuses in A. P. position show a left pansinusitis. Impression, cerebellar tumor. X-ray of skull and sinuses show pansinusitis left. Retrobulbar neuritis left." Patient was examined by Dr. Quackenboss April 14, who reported: "Right disc slightly pale with slight haziness over outer and upper portion; no engorgement of vessels; left fundus practically normal."

She had a large central adenoid and unhealthy looking tonsils with offensive secretion in crypts. The septum was deflected to the right high up. Crusty secretion both sides of nose; bulla ethmoidalis prominent on both sides. Considerable secretion beneath middle meatus. Ridge on left side in contact with middle turbinate far back. Vestibular test made by Dr. Sargent at the Eye and Ear Infirmary rather pointed to some involvement in the cerebellum. While the examination of this patient showed considerable pathology in the nose, the neurologic symptoms were so pronounced that no operation was advised or suggested. She was sent back to the Brigham Hospital, where a cerebellar exploration was done. Later developments pointed to a probable diagnosis of multiple sclerosis.

Comment.—This case is reported to emphasize the importance of not operating on cases, even with marked pathology in the nose, when the neurologic symptoms are so pronounced.

Case 40.—Dr. H. L. S., 32, referred by Dr. Verhoeff on April 14, 1922, with diagnosis of retrobulbar neuritis right. This patient being an eye, ear, nose and throat specialist, was able to give several most interesting points on his case. General health good. In 1917 the vision was 20/10 in both eyes. In 1918 he was subject to colds and intestinal disorder and had a blurring, which came on gradually in the left eye, but no attention was given to it. In September, 1920, following an acute coryza there was a central scotoma of the right eye which came on in the evening, when he found he could not read his program. Was seen by one of Dr. Sluder's assistants the following day and a double ethmoid and sphenoid exenteration was performed. The right eye cleared up and has been normal up to three weeks ago. He still has a small central scotoma in the left. Three weeks ago contracted a severe cold and has had since then a scintillating scotoma in the right eye. The peripheral vision was practically normal, but he

needed a bright light in order to read. Has had a decided
ache in the right side of the face for past few days. There
were numerous adhesions about the right sphenoid—in fact,
the cavity seemed to be entirely shut off from the nose. Rem-
nants of the middle turbinate right; considerable secretion in
the posterior portion of nose; large mass of soft polypoid tis-
sue left, in the region of the sphenoid; pharynx red.

On April 22d, under local anesthesia, the right sphenoid was
opened. The anterior wall was of bone and of about the usual
thickness. On the outer wall of the sphenoid there was a soft
mass, giving way under pressure with cotton swab, probably
edematous membrane. Fearing it might be the cavernous
sinus, it was not probed. In the posterior ethmoid region con-
siderable fibrous tissue was punched out and a small polyp
found about which there was pus. In four days the patient
reported that the scotoma had lost its brilliancy. Within three
weeks the scintillation had entirely disappeared, but there re-
mained a slight blur. Two weeks later vision was normal and
the scotoma disappeared.

Case 41.—I. G., 67, referred by Dr. Minot Davis on April
27, 1922, with diagnosis of acute retrobulbar neuritis right.
Patient in fair health but had a severe cold with pain over and
about right eye. Two weeks ago noticed change of vision
right. Things became blurry, especially in central vision.
Could see better on temporal side. Lameness of eye on move-
ment. Excessive discharge from nose. Color scotoma for
red and green; vision 2/200. Septum deflected to the right
with marked obstruction. Nose extremely narrow on that
side. On account of patient's age and rather poor general
condition it was thought best to try local treatment. Dr. Mac-
millan filmed the sinuses and reported anterior ethmoids slightly
clouded. Posterior ethmoids and sphenoid clear. Slight cloud-
ing of right antrum. Rest of sinuses negative. The sella tur-
cica was unusually small. Optic canals 5 by 5 mm. in diam-
eter. On April 29th, two days after treatment started, the
blurriness was less marked. In ten days vision fingers at fif-
teen feet. In three weeks, vision 20/40; in five weeks, 20/30
plus; on September 26th, both eyes were the same—that is,
20/30 plus.

Comment.—This case, while fairly acute with marked blocking of the nose, demonstrates how with a large optic canal it is safe to treat the patient locally. Probably many would recover under treatment as did this case, but with a small optic canal the result might not have been so favorable.

Case 42.—I. P., 60, referred by Dr. Hatch on June 27, 1922, with diagnosis of retrobulbar neuritis both. Patient has always had poor sight, but ten weeks ago there was a sudden diminution of vision, apparently without being connected with any recent infection. Noticed on awaking that he could not see as well as usual. Vision has continued to fail. Has a rather profuse secretion from nose and frequent headaches. Vision, fingers at 7 feet; fundi negative; questionable central color scotoma. Polyp right; both antra dark on transillumination; septum fairly straight; both middle turbinates large and obstructive with secretion about anterior end of right one. Dr. Macmillan filmed the accessory sinuses and reported osteoma right frontal sinus and cloudiness throughout right ethmoid labyrinth; sphenoid clear; optic canals 4 by 5 mm. Dr. H. H. Vail made a careful neurologic examination but found no evidence of any intracranial tumor. Visual fields showed central scotoma for colors and form and contraction at periphery. Patient permitted removal of polyp but refused any further operative procedure. He was treated locally for several weeks with some improvement, so that within four weeks his vision had doubled. He then dropped out of sight for nine months and on being located by the social service was reexamined. The vision had dropped back again to fingers at 7 feet, the same as when first seen. Dr. Hatch found nothing definite in the fundus except possibly a slight atrophy.

Case 43.—F. M. C., 53, referred by Dr. P. H. Thompson on June 29, 1922, with diagnosis of postneuritic optic atrophy both. Rather poor general health. About ten months ago commenced to have pain about left eye and through forehead, lasting three or four weeks. This was accompanied by marked loss of vision. Eye sensitive to pressure. Could see half a word on reading the paper. In four weeks was unable to read with left eye. Urine, blood and blood pressure negative. Vision has recently commenced to fail in the right eye and is now, with correction, 20/70; left, fingers at about 1 foot when looking straight ahead, but 5 feet laterally; both discs rather

blurred; edges pale, especially on temporal half; grayish ap-
pearance, marked on left; some swelling of left disc at upper
and outer quadrant. Visual field right showed large contrac-
tion for form and colors; small blind spot. Vision so poor in
left, field could not be taken. Dr. George X-rayed the acces-
sory sinuses and reported: "We are unable to find any posi-
tive X-ray evidence of sinus disease. Possibly there is a very
slight increase in density over right antrum and right ethmoid
cells. This change is so slight we would not consider it of
importance unless there is some clinical evidence pointing to
these regions." Both middle turbinates hypertrophied; septum
deviated to right high up and far back.

Dr. Coriat reported: "No evidence in neurologic examina-
tion which would lead me to make a diagnosis of brain tumor.
Also examined X-ray plates of Dr. George, and in addition
to findings which you already have, I noted slight mottling in
frontal region, but as there was no erosion of inner table of
skull and no widening of suture fissure or venous canals I do
not consider this of any pathologic significance."

Under general anesthesia, on July 7th, assisted by Dr. Vail,
both middle turbinates were removed and the front walls of
both sphenoids opened. The tissue on the front wall of the
sphenoids seemed very thick. Both posterior ethmoids un-
capped; no packing; rather free hemorrhage, which persisted
during afternoon and which required the use of thromboplastin
on the left side. Patient otherwise progressed favorably while
at the Infirmary and was discharged six days after the opera-
tion with improvement in vision and apparently very good
general condition. During her six days' stay at the Infirmary
she ran a normal temperature and, except the moderate bleed-
ing during the first six hours, there was no complication
whatsoever.

The patient was taken suddenly ill on July 16th, nine days
after the operation. Her family doctor told me she seemed
to be greatly prostrated, a condition which he attributed to
her heart. She failed to respond to the ordinary heart stimu-
lants and passed away within 48 hours, there being at no time
any elevation of temperature or any symptoms pointing to
meningitis or sepsis. The specimens removed by Dr. Verhoeff
were reported on as follows: "Both turbinates show an in-

creased number of fixed cells in the submucosa and slight in-
filtration of this layer with lymphoid cells. The left turbinate
in places shows submucosa converted into hyalin tissue with
stellate cells."

Comment.—This case being the only one where a death fol-
lowed, even remotely, an operation on the sinuses, seems
worthy of some comment. The eye condition was desperate,
and it was not felt that the operation was an especially hazard-
ous one. Her general condition was not of the best, but no
worse than many undergoing operations in the daily routine
of the hospital. The cause of the death is still in doubt. The
possibility of an embolus should be considered, although the
family doctor said he did not think symptoms pointed that
way. Meningitis and septicemia are out of the question. There
might have been some intracranial condition—tumor or abscess
or a cerebral hemorrhage, which latter was given in the death
certificate as the cause.

Case 44.—P. E. F., 43, referred by Dr. Walter Lancaster
on November 24, 1922, with diagnosis of retrobulbar neuritis
right. Poor health. For some months has been totally in-
capacitated. Considerable pain through head for past six
months. Eyes have felt strained and pained badly at times.
Feels as if it were about to drop out. Dizzy for last three
or four days. Subject to colds and frequent attacks of tonsil-
litis. Dr. Lancaster reported that during the past two months
patient "had developed an ill defined central scotoma especially
for colors in the right eye; also a marked tendency to spasm
of the accommodation in that eye. Has suffered from head-
aches and from a nervous breakdown. I do not believe his
scotoma is of a hysterical nature, but think a sinus is the most
likely cause. I do not believe that the eye condition is urgent
and that he may be observed with safety for a few weeks if
you consider it necessary. There is also a possibility of pitui-
tary." Vision 60/100 right. Both tonsils were rather small
but contained offensive secretion. Polypoid degeneration of
the anterior end of right middle turbinate; left middle turbi-
nate enlarged but not degenerated. Sinuses X-rayed by Dr.
Macmillan, who could find no evidence of pathology; the sella
turcica was normal; optic canals 5 by 5 mm. in diameter. Un-
der local treatment vision improved so that in two weeks it

was 60/50 and so remained. Owing to the patient's poor general condition it was not thought wise to remove the tonsils until he was somewhat improved. This was, however, done at the end of the third month. This case is still under observation.

Case 45.—E. O. A., 41, referred by Dr. H. B. C. Riemer on December 23, 1922, with diagnosis of acute retrobulbar neuritis left. Health fair; not many colds or sore throats but had a severe cold two months ago; eyes rather weak for some years; no headaches but occasional dizziness. On examination by Dr. Riemer six weeks ago, vision was normal. Eight days ago things became very blurry in left eye, which increased so that at the end of five days vision was fingers close to eye. The following day he was totally blind, not even light perception. Marked pain on rotation of eye; blurring of edges of disc and increase in capillaries of the nerve. Moderate smoker, no drugs or alcohol. Septum straight; turbinates not especially enlarged or obstructive; tonsils showed marked evidence of disease, offensive secretion removed on suction; enlargement of cervical glands; considerable adenoid tissue. Dr. Macmillan reported: "All sinuses are clear, with exception of very slight clouding of left antrum. This, however, does not suggest pus but is probably thickened membrane from some previous infection. Lower right molars both diseased. Upper left central incisor shows area of rarefication about apex suggesting abscess formation." Right optic canal 5 by 5 mm., left 4 by 5 mm. Patient had the diseased teeth extracted and tonsils removed. Left side of nose was packed with adrenalin and through a long Killian speculum the opening to the sphenoid was made out. Sinus seemed of good size and free from secretion on probing, so it was not opened. One week after the operation patient could count fingers at two feet; in three weeks, twenty feet. Two weeks after the operation Dr. Riemer reported as follows: "Left disc blurred, most pronounced on nasal half. Outline disc well defined on temporal half; numerous fine white lines radiating from disc on nasal side can be seen in retina." One month after operation vision was 20/50; disc had slight blurring, paler than normal; outline well defined; on nasal side a concentric blurring very close to nerve head. Three months after operation vision was 20/25. This patient is still under observation.

Case 46.—W. R. F., 35, referred from the Eye Clinic at the Infirmary by Dr. Worthen with diagnosis of secondary optic atrophy both. Vision 20/200. Fair general health. Five years ago eyes became blurry. Does not remember whether they were lame or painful. Treated by Dr. E. W. Clap, who gave me the following report: "First seen on April 20, 1918. Complained of blur in right eye for two weeks. Vision 20/200 in each eye, not improved by glasses. Central scotoma for green. Pupils small, equal and reacted. Temporal segment of optic nerve white and slightly cupped, edges hazy. Wassermann negative. Smoked to excess. No alcohol. Several diseased teeth. After extraction considerable improvement in vision. January 13, 1919: Vision with correction, right 20/40, left 20/70. May 22, 1919: Vision, right 20/50 plus, left 20/70, nerves unchanged. January 22, 1921: Vision 20/100 both; tension always normal; fundus unchanged. February 7, 1921: Vision 20/200 both. May 27, 1921: Right 20/200, left 20/50 plus." Some intracranial tumor was suspected when first seen by Dr. Clap and patient was referred to Dr. Clymer, but his examination was practically negative. Dr. Morrison reported that teeth showed active granuloma; sella turcica normal. There was a ridge on the right side of nose with deflection to left high up and far back. Right middle turbinate seemed to be located far back and decidedly obstructive to drainage from the posterior sinuses. Left somewhat similar. Offensive cheesy debris in tonsils. Optic canals were 5 by 5 mm. in diameter. Tonsils removed on February 27, 1923, and septum resected. March 16, 1923: Patient greatly improved in general health and can read one line lower on chart.

Comment.—This case is interesting because of the five years during which he has been under observation. Following the removal of the teeth there was marked improvement in vision, then it seemed that some other infection was producing the gradual loss and as the tonsils showed distinct infection their removal seemed to be indicated.

Case 47.—N. de F., 18, referred from the Eye Clinic of the Infirmary on March 2, 1923, by Dr. Hatch with diagnosis of retrobulbar neuritis left. Good general health. Some eye trouble two years ago when entering this country, but not a neuritis. Present trouble came on suddenly one week ago.

Noticed everything was dim when looked at with left eye.
No pain or lameness. Recovering from cold with some general headache. When first examined on March 2, 1923, vision
fingers at 3 feet; fundi normal. The nose seemed negative.
Turbinates of moderate size, no special blocking; septum
straight; tonsils large and unhealthy. X-ray of sinuses showed
cloudiness and thickened membrane lower part of left antrum;
very large sphenoid. Left optic canal 4.5 mm. in diameter;
right, the unaffected side, 5.5 mm. Following removal of the
tonsils vision improved rapidly. In one week it had gone to
fingers at 10 feet, and in three weeks was normal. The fundus
at no time showed any change.

Case 48.—B. McL., 39, referred from the Eye Clinic of
the Infirmary on March 5, 1923, by Dr. Worthen with diagnosis of chronic retrobulbar neuritis with secondary atrophy.
Slight pallor nasal side both discs; fields show scotoma; vision
10/200 both. Patient in fair health. For past ten years has
complained of defective vision both eyes. Examined in various
hospitals but no cause found for his loss of vision. Wassermann and neurologic examinations negative. Complains of
constant pain in both eyes, especially when trying to fix upon
an object. Nose appeared normal; septum straight; no enlargement or blocking from turbinates. The tonsils, although
small, were thought to be diseased. The teeth were very suspicious, and on filming them several root abscesses were discovered and a large cyst. Sinuses were negative. Optic canals
5 by 5 mm. The diseased teeth were extracted and the tonsils
removed.

Comment.—In spite of the long duration of this case it was
felt that the patient should receive the benefit naturally resulting from the removal of any purulent focus, but improvement, if any, will probably be slow. It is hoped that further
impairment of vision, at least, will not take place.

Case 49.—K. McI., 19, referred from the eye side of the
Infirmary by Dr. Lancaster on April 4, 1923, with diagnosis
of optic neuritis left. Good general health; occasional colds
and sore throats. Had a very severe cold six weeks ago with
pain over left frontal and about eye. This was accompanied
by flashes of light. Two weeks later sight failed suddenly in
the left eye so that at one time she could only see bright lights.

On admission to Infirmary on March 23, 1923, vision was 20/70— in left eye. Within a week the vision had improved to 20/40—. In the left fundus there was a mild grade optic neuritis with slight infiltration about disc. Visual fields showed contraction in outer temporal region and marked enlargement of the blind spot. The septum was deflected to left, especially far back, thus crowding the left middle turbinate; tonsils, while rather small, had some offensive secretion and a sensitive gland was found below angle of right jaw. X-ray of sinuses showed very small frontal, ethmoids clear; slight thickening of membrane lining left antrum but no evidence of pus. Optic canals were 5 by 5 mm. The patient was examined at the Neurologic Department of the Massachusetts General Hospital, where she had a lumbar puncture, but nothing abnormal was found. Wassermann negative. In view of the probable infection from the tonsils and the marked blocking of the left posterior sinuses it was deemed advisable to operate, although vision had improved considerably. The tonsils were accordingly removed; septum resected; left middle turbinate removed and a small section of the superior turbinate, which was found hanging closely over the front wall of the sphenoid. On removing this it was possible to probe the sphenoid ostium. A small amount of secretion was seen about this opening, but I did not deem it necessary to open that sinus, feeling that the correction of the deflection and the removal of the middle turbinate gave sufficient drainage and ventilation. The tonsils were found to be much worse than apparent on examination, considerable pus being removed. One month after operation, vision 20/30.

CONCLUSIONS.

The normal optic canal is practically 5.5 mm. in diameter. It varies from 3.5 to 6.5 mm. Extensive pneumatization about the canal is usually associated with narrowing. When the lesser wing above the canal is more extensive pneumatized than other regions, the canal is flattened on top. If the region beneath the canal is also pneumatized it becomes oval, while in the rather rare instances, where the bridge formed by the lateral root of the lesser wing of the sphenoid is also pneumatized, it assumes a somewhat triangular shape. Any irregularity in the contour of the canal, from whatever cause, produces a diminution in its caliber, and renders its contents more sus-

ceptible to infections from the sinuses surrounding it. It
seemed to be a fairly constant rule that the smaller the canal
the more extensive the pneumatization. It should also be em-
phasized that small canals may be round, even though sur-
rounded by pneumatic sinuses, so that the shape, while not
all important, usually indicates susceptibility to infection. The
films of the canals many times are misinterpreted, due to
faulty position. The image of the canal should always appear
in the lower outer quadrant of the orbit. While the films of
only about thirty patients with optic nerve involvement have
been studied, the conclusions from these indicate that the
smaller the canal the more the danger of permanent loss of
vision and the greater the necessity for operation. The size
of the canals is most valuable in making a differential diag-
nosis, for large canals immediately lead one to look elsewhere
than in the accessory sinuses for the cause of the amblyopia.
This has already proved of great value in two recent cases—
one due to multiple sclerosis and the other to an enlarged
pituitary, although both cases had marked pathology in the
nose. If future cases substantiate the findings in those already
studied, it will mean that a canal of 4 mm. or less, in a case
of severe optic nerve involvement, indicates the necessity for
immediate ventilation of the posterior sinuses to prevent per-
manent atrophy, unless some other definite focus can be found.
A 4.5 mm. canal gives greater leeway for study and investi-
gation. Optic atrophy is less to be feared. A 5 mm. canal
would probably recover from almost any acute attack, either
spontaneously or under local treatment. Then if some focus
of infection is found, diseased teeth or tonsils, for instance,
it should be removed as a preventive to recurrences.

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NORMAL

Sphenoid top of
lesser wing.

Roof of
right orbit.

Frontal sinus.

Outer rim
of orbit.

Optic foramen.

Posterior limit.

Sphenoid Zygo-
matic arch

Ethmoid cells.

Roof of antrum.
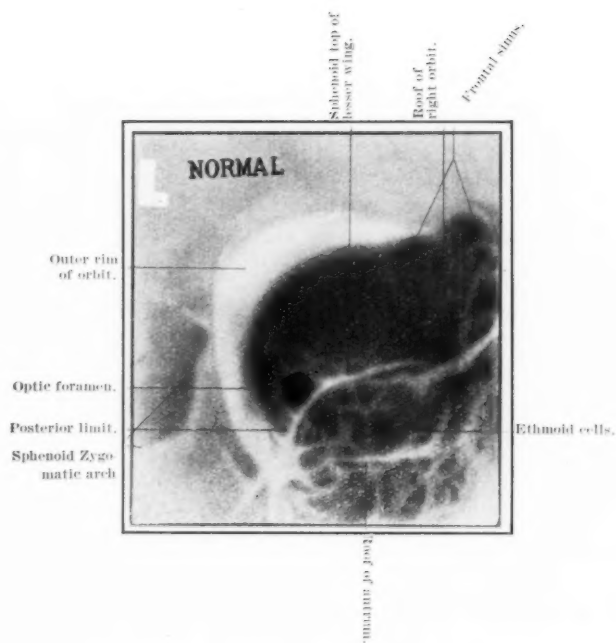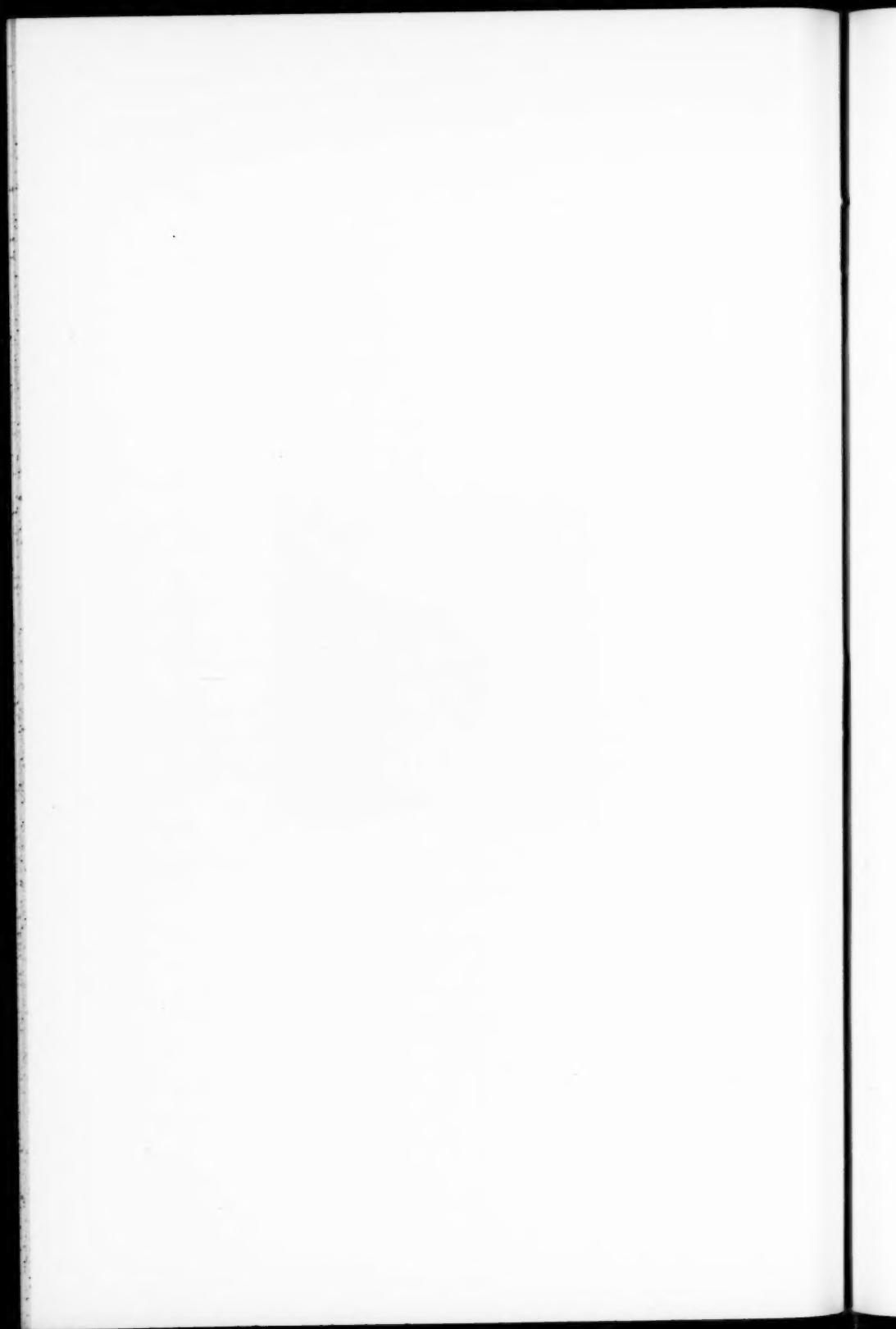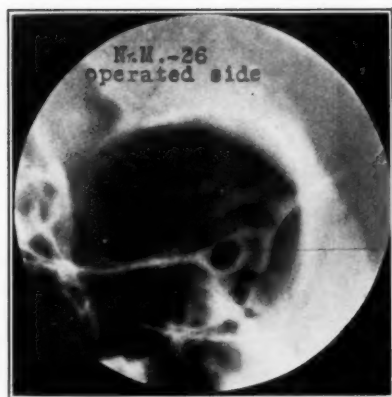
Plate 1.

Optic canal.

Plate 2.

Shows an oval canal in the correct position, measuring 4x55. Practically normal vision following opening the posterior sinuses.
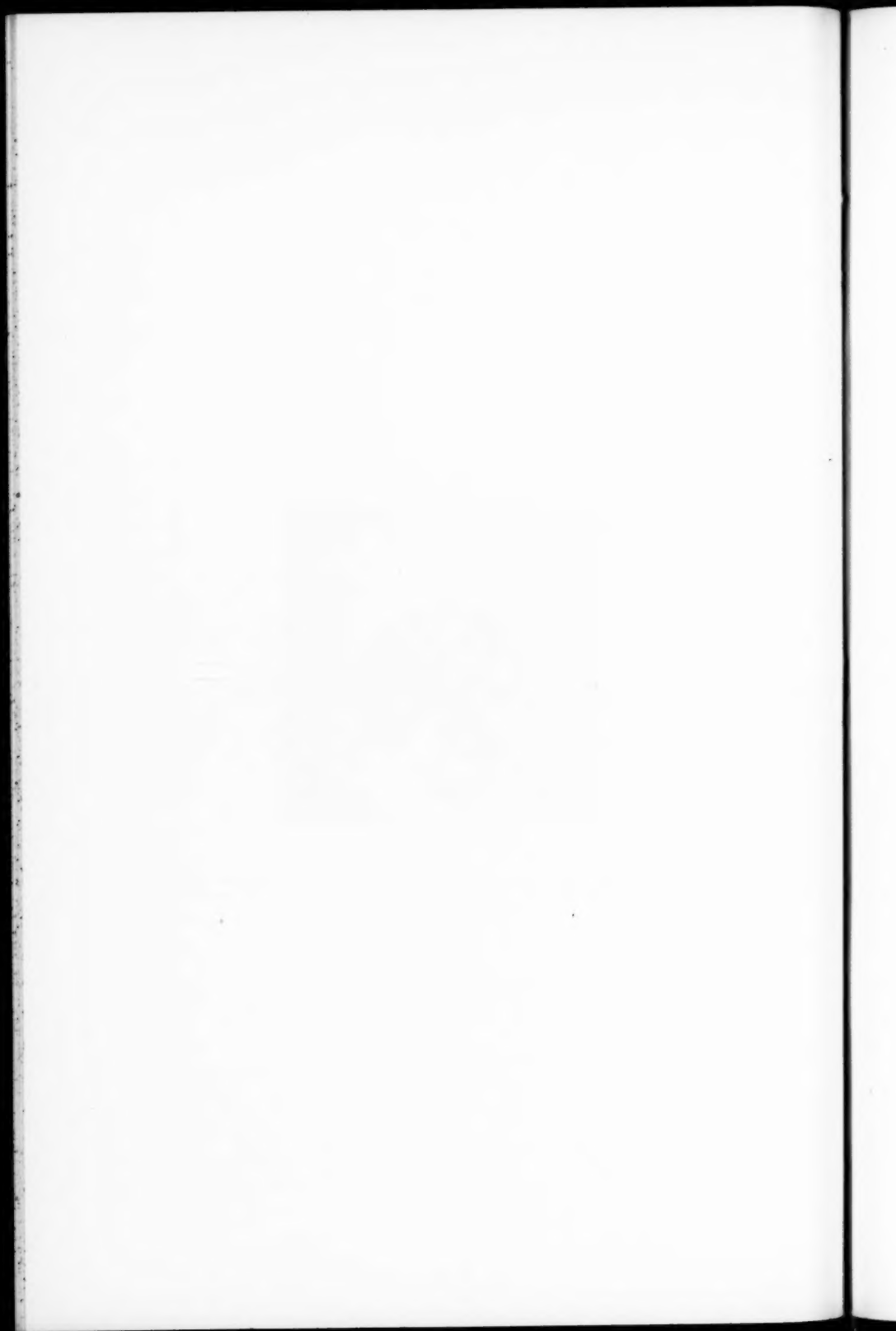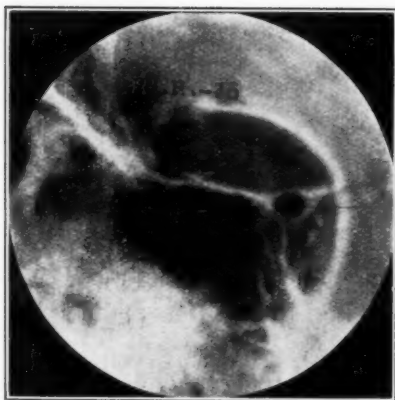
Optic canal.

Plate 3.

Shows a normal canal rather high in the orbit but it compares well
with the next plate of the other eye.
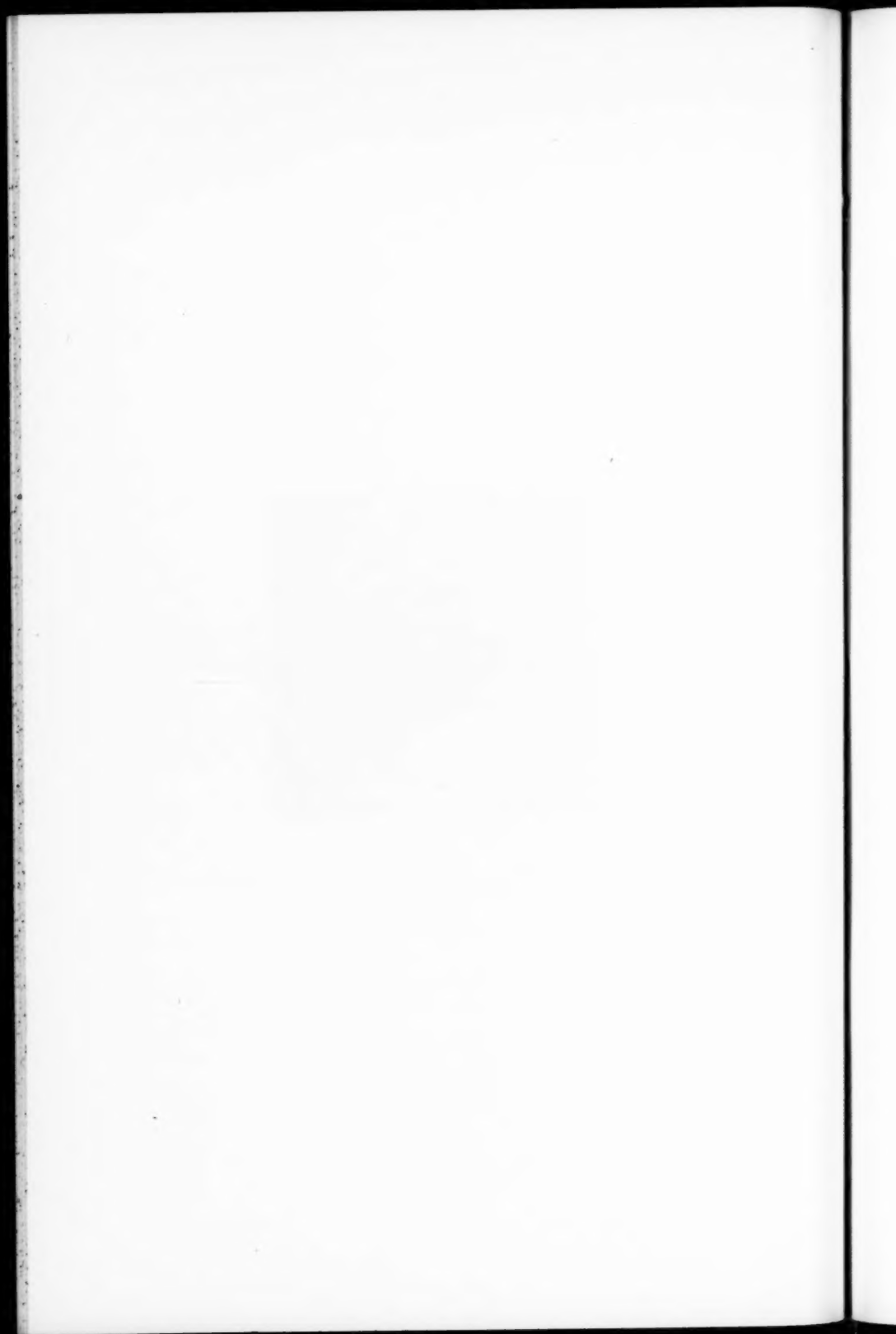
Optic canal.
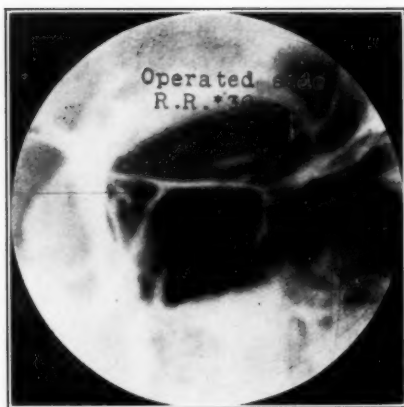
Plate 4.

Shows a small oval canal measuring 4x45 mm. Severe neuritis
relieved by opening posterior sinuses.
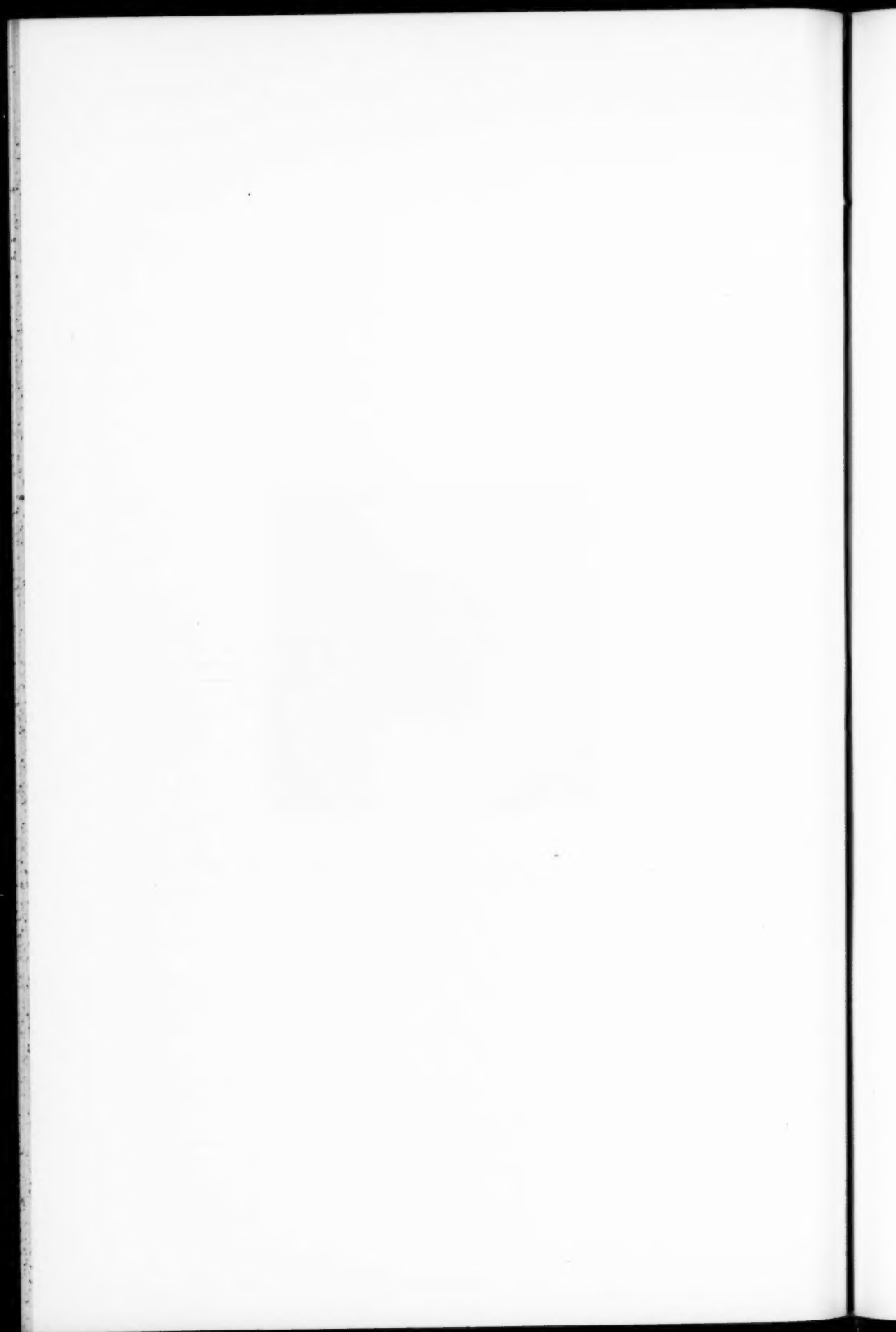
Optic canal.

Plate 5.

Smallest canal measured 3.5x4 mm. Permanent optic atrophy
following acute neuritis. No operation.
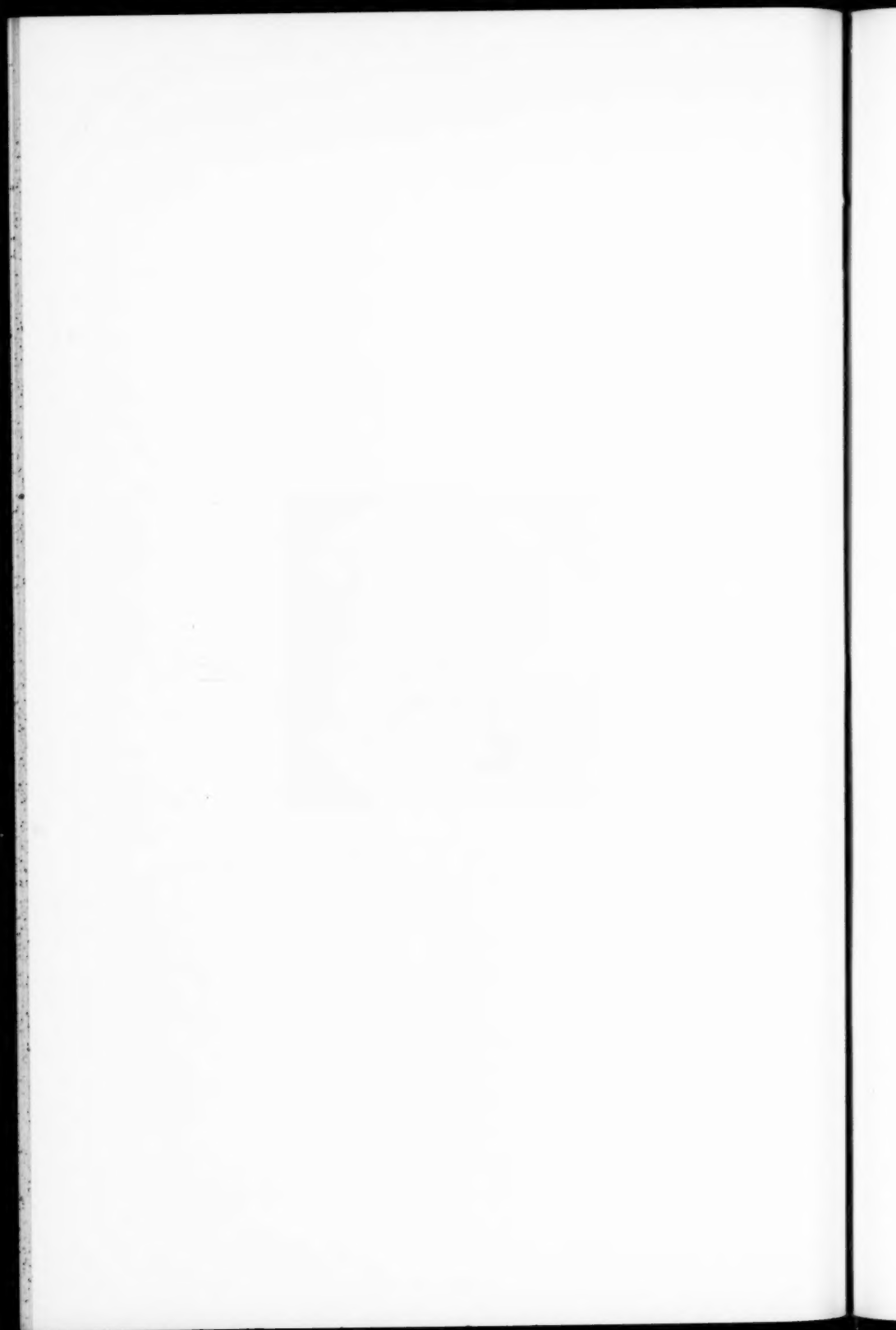
IV.

NASOPHARYNGOSCOPY UNDER NEGATIVE PRESSURE.*

By R. P. Scholz, M. D.,

St. Louis.

To make easier of diagnosis a certain group of cases in rhinology, and to facilitate the localization of diseased foci in certain others, we have experimented with combined application of nasal endoscopy and intranasal suction.

Since the introduction of intranasal negative pressure, as a means of diagnosis in rhinology by Travalgyre in the year 1900,[1] it has been used with more or less uncertainty. There has always been considerable doubt as to its true value because of the many possibilities of error in the technic. Nevertheless, during the past fourteen years, we have found intranasal suction a helpful adjunct in diagnosis of especially the obscure suppurative ethmoid diseases,[2] but always there has remained a doubt as to whether the pathologic secretion sucked into view actually came from one of the cells or whether it had merely been drawn from a recess of the nasal chamber. With the combined use of these two diagnostic means, intranasal suction, as used by Wagers[3] and others, and nasal endoscopy, as set forth by the late Holmes,[4] one is enabled actually to see from whence the pathologic material comes. This does away with any doubt as to its source, and furthermore enables one to observe the behavior of the nasal tissues during the application of nasal pressure.

In my experiments I began with the very crude application of suction and endoscopy, applying both instruments in the same side of the nose. This I soon found cumbersome, and therefore resorted to the application of the endoscope in the side of the nose to be examined, applying the suction tip in the other side. This method was fairly practical, but was

discarded for a more simple technic, made possible by the creation of a modified nasal olive, through which the nasopharyngoscope could be freely passed and manipulated.

The instrument consists of a modified conical metallic tip or capsule, 4.5 cm. long, its distal end compressed so as to be 1 cm. laterally and 1.6 cm. vertically in cross section, near the apex. From this to the proximal end the instrument increases in size, gradually becoming circular, until it attains a diameter of 2.6 cm. The proximal end is fitted with a removable screw cap, in the center of which there is an adjustable valve, through which an ordinary Holmes nasopharyngoscope can be passed, to traverse the length of the capsule, emerging from the distal end. Leading into the lower side of the capsule near the proximal end is the air lead or hose attachment, and at the apex, just above the outlet through which the endoscope passes, is the air inlet.

The size and construction of this suction tip is such that it fits approximately either side of the nose and can be used interchangeably through the right or left nostril. Its lateral compression and dimensions give it a decided advantage over most nasal olives in that it fits through the collapsible vestibule and well beyond into the nasal chamber proper.[5] The fact that the air intake is at the apex of the instrument and above the opening for the rhinoscope places it well away from the soft tissues of the inferior turbinate, thereby overcoming the objection which many might have to its construction.

The electric connection to the endoscope is made through a rheostat in the usual way. The suction tip is attached to a (Victor) suction and release pump by means of a soft rubber hose, the walls of which have sufficient resistance to retain its lumen during suction. A secretion reservoir intervenes between the instrument and the pump.

Coffin[6] also found interrupted negative air pressure more effective and less harmful to the tissues than continuous suction. I wish to emphasize that only mild suction should be used to begin with, and by that I mean just sufficient to hold up the elevated palate without causing pain or discomfort to the patient. Greater force is unnecessary and less efficient but harmful. Since the quantity and degree of suction tolerated by certain individuals and individual tissues

vary, one must cautiously feel his way by increasing the force, using as his guide the patient's expression of pain or discomfort. I have not found the manometer of Horn[7] clinically practical. Mild suction makes possible the detection of the finer tissue changes.

Levenger states that with suction the tissues become so engorged that they fill the entire nasal space. This we have not found to be the case, except where violent suction is applied over a longer period.

The examination is made in a dark room. The patient is seated in a comfortable chair; the head supported by an attendant or head rest. The operator sits before the patient on a stool or stands if he prefers. To produce shrinkage and anesthesia of the nasal mucosa, a solution of 2 per cent cocain, to a dram of which a drop of 1/2,000 adrenalin has been added, is applied to the nasal passage to be examined. The other side of the nose, which is not so treated, retains its sensation and serves as an indicator for the quantity of negative pressure the patient is able to tolerate without pain. Where both sides of the nose are to be examined, only partial anesthesia should be produced in the two sides of the nose, so as partly to retain pain sense. Next the nose is cleansed of secretion, by the patient gently blowing first one then the other side with the head bent forward. If necessary the examiner can cleanse it by means of swabs.

A preliminary examination should then be made of the nasal cavity, preferably with the rhinoscope, so as to be certain that all secretion has been cleansed from the nasal cavity proper. Before introducing the instrument it is best to instruct the patient as to what he is to expect, namely, that the examination is not to be painful, and as pain is one of the examiner's guides he should let its presence be known.

With the nasopharyngoscope, passed through the suction tip and protruding about 3 cm. beyond its apex, the light burning and intermittent suction in action, the instrument is introduced into the nose. This is best accomplished by the examiner slightly tilting up the tip of the nose, with the thumb of his left hand, and passing the instrument into the nose with the other. The tip of the endoscope should follow along the

floor of the nose, as Holmes directed,[*] with the light and lens
pointing upward so as to have visible the upper landmarks of
the nose, and also to avoid besmearing the lamp and fenestra
with secretion, which would be apt to occur if the scope were
otherwise directed. After the introduction of the instrument
in the above fashion, the suction tip is introduced by sliding
it along the endoscope until it passes through the vestibule
into the nose as far as possible. The nasal tip is then grasped
by the thumb and forefinger of the left hand, and the nose
compressed about it with the second and third fingers of the
same hand, the right hand of the operator remaining free to
manipulate the endoscope. Further placing of the instrument
is directed through the endoscope.

When it is suitably placed for examination, the patient is
instructed to close off the pharyx with his soft palate by swal-
lowing, or by repeating the sound "K-K-K-" or "hook-hook-
hook-," whereupon the soft palate is held up by the force of
the suction. In the beginning the suction should be only suf-
ficient to hold up the soft palate, but by increasing it cau-
tiously the pain stage or stage of discomfort may always be
avoided. Walb[9] concluded that the force should never exceed
8/10 cm. of mercury in an acutely inflamed case, or 12/17
cm. in a chronic case.

The operator is then prepared to proceed with the examina-
tion. The soft rubber bushing in the valve of the nasal tip
permits of free rotation, retraction or deeper introduction of
the endoscope. These, together with depression, elevation or
the lateral motions of the external end of the instrument,
permit of a broad latitude of direction.

The main portion of the nose and nasopharynx can then
readily be inspected. The roomier the nose, the easier is the
examination, and this is true especially in the atrophic cases,
or cases from which much tissue has been lost from a patho-
logic process, or where the middle turbinate has been removed
partially or in toto. Easier still are those where much exen-
teration of tissue has been previously undertaken, as is the
case after an exenteration of the ethmoid labyrinth. As is well
known, what at the time appears to the operator as a sat-
isfactory and complete exenteration of the ethmoid labyrinth

may be incomplete, due to the overlooking of an obscure and remotely placed cell, which later gives rise to trouble, necessitating future interference.

In a large percentage of cases the endoscope can be used to examine the regions under the inferior turbinate, under the middle turbinates or the olfactory fissure, much as Killian[1] used his long bladed speculum. By cautiously applying force, we are enabled to squeeze the rounded end of the endoscope into the above mentioned spaces. Killian found that the thin turbinates, especially the middle one, and the upper portion of the nasal septum were flexible to the extent of permitting them to be crowded aside sufficiently to introduce an instrument such as his long bladed speculum. Since the closed blades of Killian's instrument measure 3 cm. near the tip, and the caliber of the Holmes endoscope but 4 cm., almost any recess of the nose into which the Killian instrument can be passed is also accessible by the latter. Almost every recess of the nose can be inspected by the rhinoscope during the application of negative pressure.

In the examination of the olfactory fissure, it is best to introduce the instrument between the septum and the middle turbinate anteriorly, and proceed back until the end comes in contact with the anterior surface of the body of the sphenoid, then descend, but not so low as to allow the middle turbinate to close in over the instrument. Then by retracting, the region of the cribriform plate can be examined. Rotation inward brings the septum into view; rotation outward, the inner surface of the middle turbinate anteriorly; posteriorly, the superior turbinate and superior meatus, with the orifices of the posterior ethmoid cells come into view. The middle meatus is examined much in the same manner. The endoscope is passed between the anterior end of the middle turbinate and the lateral wall of the nose, and well back, and as low as the middle turbinate will permit, without slipping over the endoscope. Rotation inward brings to view the under surface of the middle turbinate; upward, its attachment; outward, the interesting lateral wall in which can be seen the hiatus semilunaris, bulla ethmoidalis, ostium maxillæ and several of the foramina of the anterior ethmoid cells, etc. The scope usually can be passed under the inferior turbinate, so that the nasal

end of the lacrimal duct can readily be examined as well as the entire inferior meatus.

In examining these narrow regions, much difficulty will at first be encountered. Because of the proximity of the lens to the objects examined a confusing picture will present itself, the parts being so greatly magnified. Annoyance may also be met with in keeping the lamp and fenestra clean of secretion and blood.

It goes without saying that great care must be exercised in the use of the endoscope, when guiding it into these narrow recesses, lest too great force cause traumatism. Especially should we avoid laceration of the mucosa, for hemorrhage is a disturbing factor in this form of examination.

Some very interesting tissue changes are noted when mild suction is applied. First, the tissue takes on an intensity of color, from light shading gradually to a deeper pink, and finally red, with increasing bulk (both of these changes are due to vascular engorgement). As the intermittent suction continues one sees a rhythmic pulsation, synchronous with the intermission of the suction, in all redundant tissue and larger blood vessels.

It appears to the writer, although not positively confirmed, that areas slightly congested before the application of suction, as in the case of tissue mildly inflamed or tissue about an inflamed focus, take on this suction congestion a little sooner and a little more intensely than the normal mucous membrane, making possible the detection of mild inflammation of the mucous membrane of the nose, about the orifice of diseased sinuses, etc. This phenomenon has not been noticed in the chronic areas of the inflammation.

The cavernous tissue, especially vascular hypertrophies, soon balloon up, vividly pulsating, as already described. The ordinary fibrous hypertrophy, as seen at the posterior end and inferior margin of the inferior turbinate, and at the inferior end of the middle turbinate, increase in volume and pulsate, perceptibly. Edematous and fibro-edematous tissue show the above mentioned vascular engorgement distinctly when suction is applied. Then, too, we have repeatedly seen swollen and edematous tissue about the orifice of an inflamed sinus drawn into

view as a puckering out of the tissue, or a tiny pedunculated polyp actually drawn from a diseased sinus into the nasal cavity. Pathologic secretion such as pus has been seen to appear in the orifice, or to be drawn out of a diseased cell into the nasal cavity. Where there is a large quantity of secretion, a portion of it usually remains in the nasal cavity after suction is released, but not so with smaller quantities. These usually return to the cell due to the counter suction, which fact has been pointed out by Smith.[11] Upon subsequent inspection of the nose, pus will not be found.

Tilting the head, so that the orifice of the examined cavity is brought to the most dependent portion of the cell, greatly facilitates the evacuation of secretion, as was pointed out by Suan.[12] Others have found that this evacuation of secretion need not be due to actual force of suction upon it, but more often to the lining membrane of the bony sinus taking on bulk, brought on by congestion, thereby lessening the cell capacity and forcing out its contents. With release of the suction and the passing of the engorgement there is a tendency to vacuum formation which draws back the secretion into the cell.

Not in every case is the application of this instrument practical or possible, but where it can be used it greatly aids in diagnosis. When the Holmes nasopharyngoscope cannot be used or where suction is contraindicated, this form of examination is impossible. While until now we have worked with but one sized suction tip, several others of varying sizes will be constructed to fit the nose of persons of varying ages, although this one in use, because of its conical form, is suitable for almost all adults and many young individuals.

In conclusion, I wish to state that I believe that the combination of the nasal suction and rhinoscopy by the above method gives added value to either or both of these means, enabling the operator to make a more exact intranasal examination. It makes possible the diagnosis of remote and obscure diseased fossæ, otherwise extremely difficult to localize, by drawing into view secretions or pathologic tissue from the areas whereby we are enabled to make a diagnosis. It gives us the means of detecting mild circumscribed inflammation of the mucosa which otherwise might escape notice. Often

this mild inflammation of the mucosa enables us to detect deeper inflammation in the tissue or in the neighboring sinuses. It is our belief that further study of the eustachian tube by this method will be of additional diagnostic value.

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PLATE I.
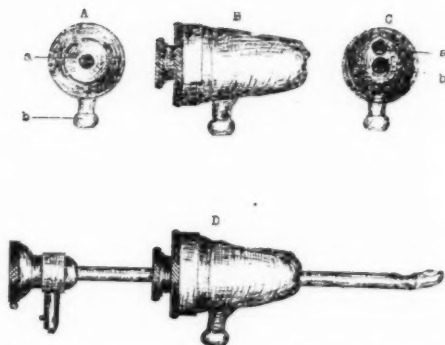
A—Proximal end of nasal tip. (a)—Opening for nasoscope; (b) Air intake. B—Lateral view of nasal tip. C—Distal end of nasal tip. (a) Air outlet; (b) Outlet for nasoscope. D—Nasal tip with nasoscope inserted.
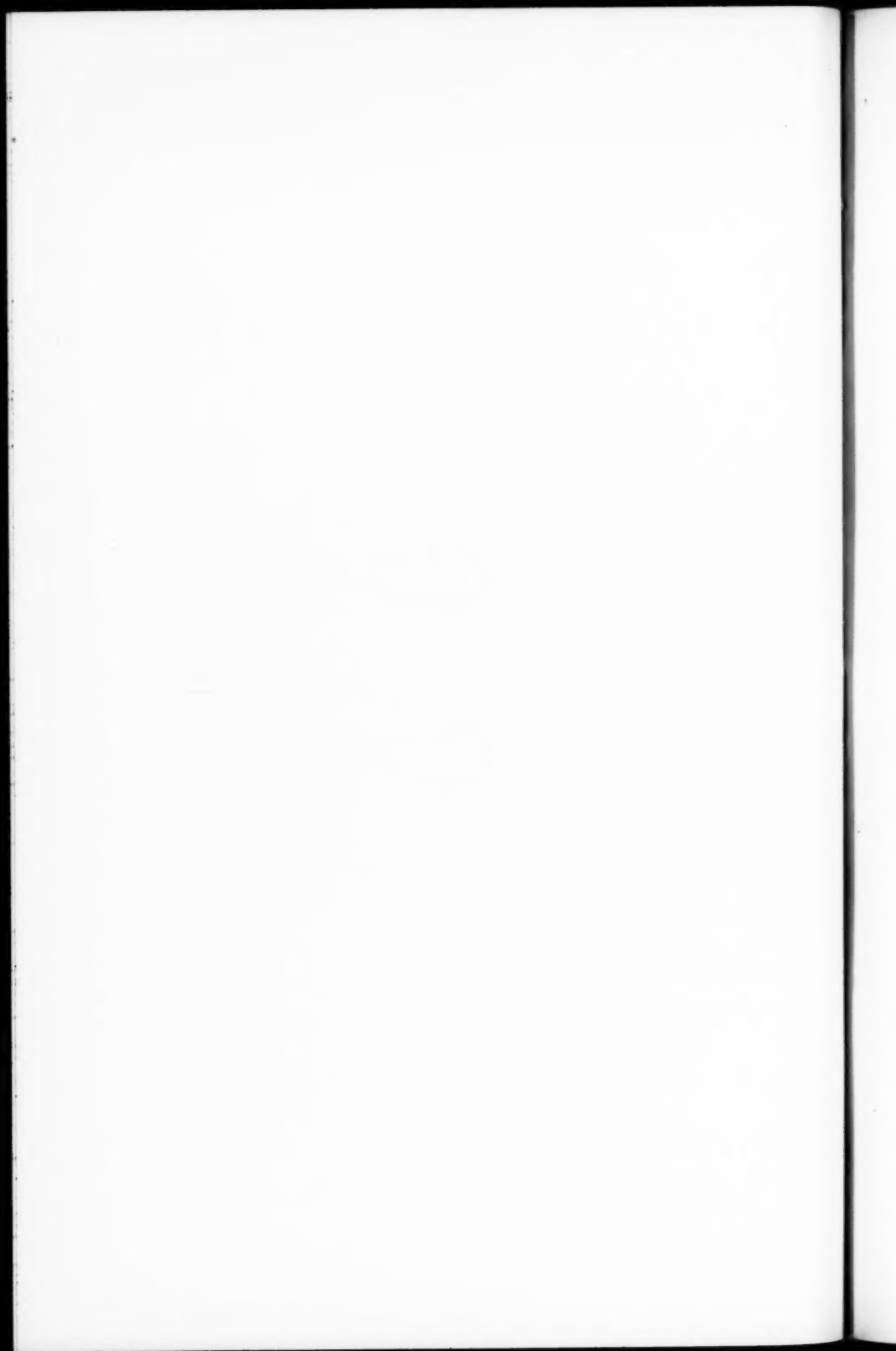
PLATE II.

The nasal tip with the inserted rhinoscope, applied in the patient's nose. The instrument is held by the thumb and forefinger, and the nose compressed about it with the second and third fingers of the operator's left hand, the right hand remaining free to manipulate the nasopharyngoscope in the usual way.
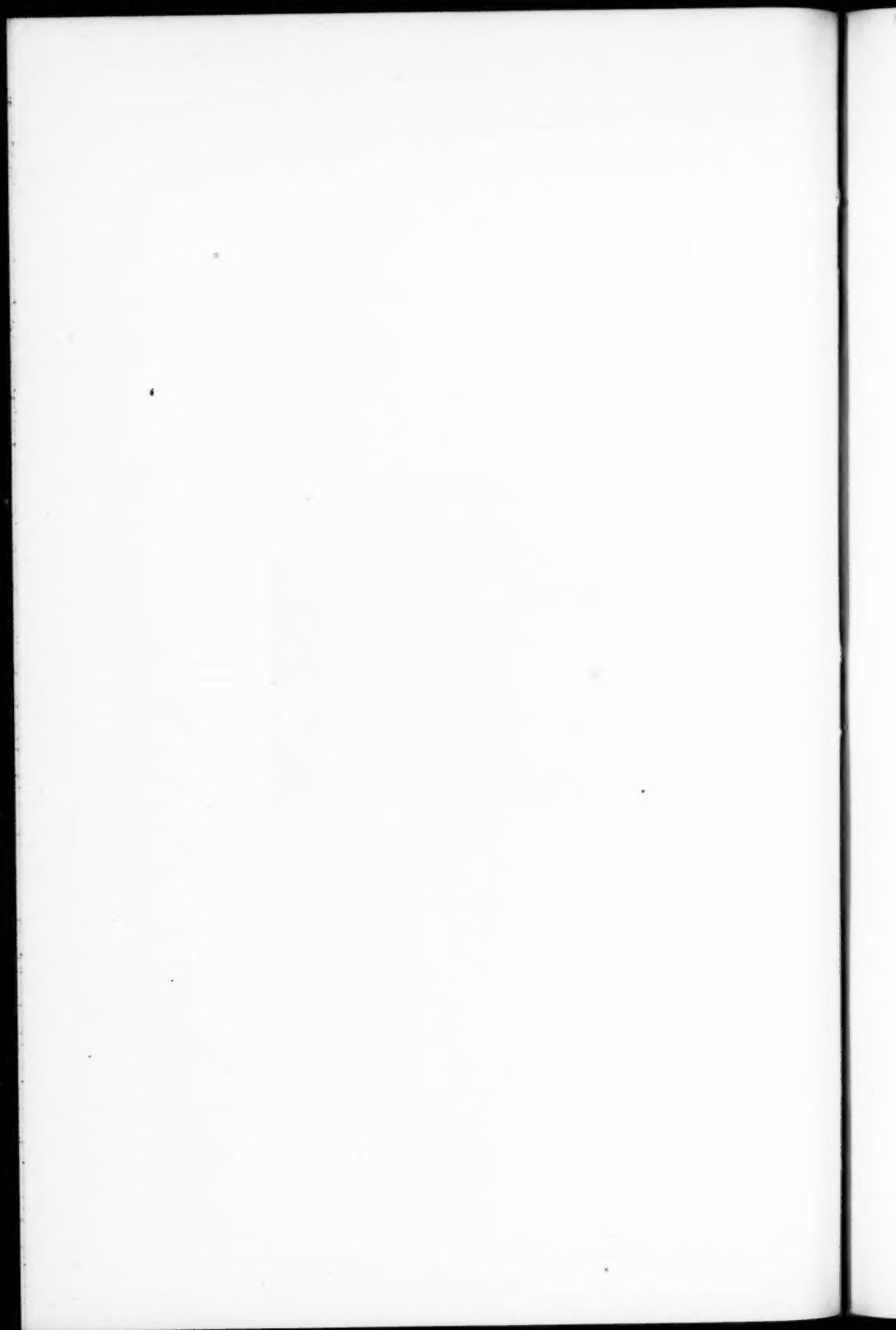
THE DIAGNOSIS, DIFFERENTIAL DIAGNOSIS AND PROGNOSIS OF LARYNGEAL TUBERCULOSIS.*

BY FRANK R. SPENCER, A. B., M. D.

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Laryngeal tuberculosis is such a frequent complication of pulmonary tuberculosis that it assumes great importance in the practice of laryngology and should, therefore, be discussed in laryngologic meetings more frequently than it is. I am particularly interested in the early diagnosis, because it often means a cure or, at least, an arrest of the disease. Many authors—e. g., Minor,[1] Curtis,[2] Thompson,[3] Carmody,[4] Grayson,[5] Ellis in discussing Freudenthal's[6] paper, and others have laid stress upon the value of an early diagnosis. Greene[7] says, "A well equipped sanatorium, with a treatment room for throat conditions, is almost a requisite for the proper care of laryngeal tuberculosis." I would add to this a well trained laryngologist. Levy[8] remarks, "No subject in medicine possesses a more extensive or interesting history than tuberculosis, nor can any disease boast of more attempts at solving the problem of its treatment with less permanent, definite and satisfactory results." Dworetzky[9] adds, "The importance of the early diagnosis of pulmonary tuberculosis has been fully established. The early detection of the pulmonary lesion has in the majority of instances enabled us either to cure the lesion or at least check its progress.

"The early diagnosis of laryngeal tuberculosis, however, is still being neglected, and too often we meet the advanced laryngeal cases, in which conservative treatment is of no avail, while radical treatment is also useless because of the existing extensive destruction of tissue. Such involvement of the larynx could often be avoided if treatment were instituted in the early infiltrative stage. Thus it could safely be said that, as in pulmonary tuberculosis, the prognosis and the successful

treatment of tuberculous laryngitis depends largely on an early diagnosis."

The primary type is so rare, if, indeed, it exists at all, that it scarcely deserves serious consideration. My only excuse for mentioning it is that we occasionally hear of the primary form. Arrowsmith[10] says, "It is conceded today that primary laryngeal tuberculosis probably never occurs and that most individuals who exhibit tuberculous manifestations in mature life have been infected in childhood or infancy, the infection remaining latent as a rule, until some adventitious stimulus spurs it into activity in any or all of its protean forms." Wood,[11] however, believes that it is unquestionable that primary tuberculosis of the larynx has occurred, though it is exceedingly rare.

Many laryngologists of wide experience, such as Dr. Robert Levy, state that the primary type of the disease has almost never been encountered in their many years of experience in the diagnosis of laryngeal tuberculosis. Hastings[12] says, "Tuberculosis of the larynx is rarely, if ever, primary. The primary focus may be undemonstrable except by autopsy. In view of the numerous positive tuberculin test observations and confirmatory autopsy records in apparently nontuberculous patients a diagnosis of primary tuberculosis of the larynx can well be questioned. Certainly a verdict of 'not proven,' so far as its being primary is legitimate, until a searching autopsy is made." Levy made a similar statement in discussing Hurd's[13] paper. Sir St. Clair Thomson[14] says, "Primary tuberculosis of the larynx, as shown by postmortem integrity of the lungs, is so extremely rare that for practical purposes its possibility may be neglected. When cases are met with where no pulmonary lesion can be detected, this is simply because our present methods of investigation are inadequate to detect early or limited deposits in the chest."

Freudenthal[15] believes, "Primary tuberculosis of the larynx does occur, and more frequently than has hitherto been accepted." Chappel[16] expressed a similar opinion.

Dworetzky[17] says, "It can readily be seen that a thorough knowledge of clinical pathology is fundamental. Without a full conception of the various phases and course of a lesion, we are scientifically unable to prognosticate and treat a disease."

The following classification based on clinical pathology includes almost all cases unless complicated by other diseases:

I. Primary. Extremely rare if it exists at all.

II. Secondary. Types based on macroscopic pathology.
    1. Vascular changes.
        a. Anemia.
        b. Hyperemia.
    2. Infiltration.
    3. Tuberculoma.
    4. Ulceration.
        a. Acute or active.
        b. Chronic or sluggish.
        c. Superficial.
        d. Deep.
    5. Edema.
    6. Fibrosis.
    7. Perichondritis.
    8. Chondritis.
    9. Necrosis.

III. Duration.
    1. Acute.
    2. Subacute.
    3. Chronic.

IV. Stages.
    1. Stage I.
    2. Stage II.
    3. Stage III.

On a former occasion the writer[18] gave a briefer classification, but the present one is more complete.

Dworetzky[11] advocates the following classification:

"Peracute tuberculosis of the larynx.

"Acute tuberculosis of the larynx—Stage 1, Stage 2, Stage 3.

"Subacute tuberculosis of the larynx—Stage 1, Stage 2, Stage 3.

"Chronic tuberculosis of the larynx—Stage 1, Stage 2, Stage 3."

While acute laryngeal tuberculosis is rare, it may and does occur. It may complicate any stage of either the laryngeal or pulmonary disease. It is usually seen in patients who have very little, if any, resistance to pulmonary tuberculosis and

who rapidly succumb. It is particularly this type of disease which has been called by the laity "hasty," "quick," or "galloping" consumption, and it has made physicians speak of the laryngeal complication as the "death warrant." Patients who have suffered for years from pulmonary tuberculosis of a mild type may lose what resistance they have had and suddenly develop a rapidly fatal acute tuberculous laryngitis.

The subacute type is more frequent than the acute and will usually pass into the chronic stage under proper treatment for the pulmonary and laryngeal disease. Such patients suffer from hoarseness or even aphonia, and the larynx shows moderate involvement, particularly of the posterior wall. Infiltration, tuberculomata, or edema may be present, but rarely ulceration.

The chronic type is the one most frequently found. There is infiltration with a decided tendency to fibrosis. The patient may complain of a dry throat and hoarseness. If the infiltration is limited to the interarytenoid sulcus the disease may be easily overlooked. Fortunately, the disease may show very little tendency to extend to the other parts of the larynx. This type, therefore, offers the best prognosis and under proper treatment the best opportunity for a cure.

Stages one, two and three refer to the amount of laryngeal involvement found upon examination. Stage one means slight or limited involvement, and stage three extensive involvement of all or nearly all parts of the larynx. Stage two indicates an intermediate involvement.

One of the very first things a patient will complain about, in the early involvement of the larynx, is a slight huskiness of the voice. This may be present in the morning upon getting up and may be relieved by coughing or clearing the throat, only to return later in the day if the patient becomes tired or is allowed to talk. At this early stage an examination may reveal an anemia or a slight hyperemia, but at most the laryngeal changes from the normal are not marked. If the warning which an early laryngitis gives is not heeded, the hoarseness may become more noticeable in the morning and may persist all day. This is to be expected if there is an early infiltration over the posterior wall. The patient is fully aware that he has

to make a greater effort to talk than formerly. Bane[19] says that vigorous efforts should be put forth to check the local disease in the early stages.

Sollenberger[20] thinks that "mere weakening of the voice, vocal fatigue on talking, hoarseness, a sense of dryness in the throat, troublesome localized tickling, a lump-like tickling on swallowing, pain, a feeling of languor or debility and increased dry cough" are all important in the early stage of the laryngeal complication. He furthermore says, "These signs no doubt often herald the approach of the disease and should always be an indication for speedy laryngoscopic examination. But they are also signs of actual involvement. Sometimes nearly all these external signs are absent in the process of involvement, even to the point of disintegration."

The laryngeal disease is by no means continuously progressive. Weeks and months may go by with very little change in the laryngeal picture, especially if the pulmonary disease is improving. That the larynx sometimes heals spontaneously in this very early stage there can be very little doubt. Levy[24] has recently mentioned this again. On the other hand, if the pulmonary tuberculosis is rapidly extending, the laryngeal disease will usually increase too. Dean[25] and Mullin[26] have shown the desirability of cooperation between the laryngologist and internist if the best results are to be obtained for the patient.

With more extensive involvement of the larynx there is likely to be almost constant hoarseness, and the throat feels dry. There may be tickling, stinging, fullness, cough and expectoration. Examination may reveal infiltration over the posterior laryngeal wall. This is due to sputum remaining in contact with the posterior half of the larynx, for hours at a time during sleep, if we accept the older theory of direct infection with the sputum, as advanced by Louis.[27] It is difficult to explain the early and frequent involvement of the posterior wall in any other way. Dworetzky[28] found that 91 per cent of his cases of laryngeal tuberculosis had positive sputa. The posterior wall, however, is subjected to more trauma during paroxysms of coughing than the anterior. Minor[21] has recently shown many of the early manifestations involving the larynx. Infection through the lymph and blood stream, as

advocated by Heinze, is just as probable, especially with involvement of the anterior half of the larynx.

If the infiltration becomes more extensive the lateral walls, the arytenoids, the true and false cords, or even the epiglottis, may be involved. Thomson[14] says, "Different parts of the larynx are usually involved in the following order:

(1) The arytenoids, (2) the interarytenoid region, (3) the vocal cords, (4) the ventricular bands, (5) the epiglottis."

Kyle[22] says, "The epiglottis is a favorite site for tuberculous infiltration, and this organ may assume a simple globular, puffed form, a thickened crescentic shape, or simulate the Turkish turban—the socalled 'turban' epiglottis." He also mentions the importance of the "club shaped" arytenoids when the latter are infiltrated. Levy[23] says, "In that form of laryngeal tuberculosis in which there is little or no tendency to ulceration, the form characterized by more or less firm infiltration, the progress of the general disease is not materially affected."

Slight elevations above the surface, due to tubercles or tuberculomata, may occur with the infiltration or may follow the infiltrative stage. These may have a smooth exterior, but are usually uneven and resemble a papilloma. In fact, a tuberculoma is frequently mistaken for a papilloma. Trautman[29] says that tuberculomata are among the earliest manifestations. If these obstruct the lumen of the larynx very much, the patient will have aphonia, dyspnea, air hunger and dysphagia. Tubercles, at any time or in any stage of the disease, interfere with the blood and lymph supply to that part of the larynx. This leads subsequently to ulceration and edema.

Tuberculous ulcers, in the beginning, are usually superficial and have "mouse eaten or mouse nibbled edges." They are covered with a thin layer of pus or sputum. This can be sprayed off reasonably well with any alkalin antiseptic and the ulcers stained with a spray of 2 per cent fluorescein in one-half of 1 per cent aqueous solution of sodium bicarbonate. Later in the disease the ulcers may be deep, but they do not have the sharply defined, "punched out" edges so often seen in lues. While they may be acute or active, if the disease is rapidly progressive, they are far more often chronic or sluggish. Dysphagia, or even odynphagia, is almost certain to be

the most harassing subjective symptom of ulceration, and this applies particularly to the acute ulcers. Odynphagia, if not relieved, is followed by secondary anemia, emaciation and a rapidly fatal termination.

The preceding picture is by no means always so typical, especially when lues and tuberculosis exist together. Carmody[1] says, "When there is much scar tissue we may and do frequently have syphilitic infection also." Grünwald[30] has emphasized the importance of watching for lesions in the larynx due to syphilis and tuberculosis. He shows colored plates in his textbook which illustrate the dual infections. Mixed infections, which are so common in pulmonary tuberculosis, also mask the objective signs and symptoms.

Fetterolf[31] classifies the ulcerative stages as follows: "(a) Infiltration with superficial ulceration, (b) infiltration and deep ulceration, and (c) infiltration with superficial and deep ulceration." This classification has much in its favor, because we know that frequently the ulcers form on an infiltrated base.

Edema may be an early symptom, but it usually occurs late. It is often designated as "ashen gray" when seen in laryngeal tuberculosis. Personally, I always feel less optimistic regarding the prognosis when there is extensive edema of the larynx, because it interferes seriously with the lymph and blood supply to that portion of the larynx. Lockard's[32] textbook illustrates the edema well, and his[33] more recent article is decidedly worth reading. He has[34] shown microscopically that the necrosis of the cartilage of the epiglottis may be due entirely to tubercle bacilli, and that it is not necessarily dependent upon a secondary mixed infection. If there is only a very limited involvement fibroid changes may take place slowly, followed by very little change in the clinical picture for months. However, perichondritis, chondritis and necrosis are apt to follow the edema in rapid succession with a fatal termination. The patients suffer from the secondary anemia, emaciation, severe odynphagia, high fever, night sweats, dyspnea, anorexia, etc. The laryngeal landmarks are obliterated and the lumen decidedly narrowed, so that tracheotomy may be required. Thomson[14] says, "Perichondritis of the thyroid may produce a swelling of the ventricular band if the inner surface is attacked. The purulent collection makes its way into the

larynx. The external surface of the thyroid plate may also be affected. A swelling then takes place externally, which is not inflamed and is seldom painful. An incision into it yields pus and leads down to denuded cartilage." Dworetzky[35] adds, "The peracute cases of laryngeal tuberculosis, fortunately, are infrequent, compared with any of the other forms. These cases are very rarely seen in the stage of edema, but usually present extensive ulceration and perichondritis. They are often accompanied by tuberculosis of the faucial tonsils and soft palate. Dysphagia and aphonia are always present, especially when the pharynx is involved. Emaciation is rapid; the patient is febrile and soon becomes moribund. Other organs of the body, such as the intestines and meninges, are often also involved. The peracute type usually begins as such, although occasionally it follows the acute type."

Syphilis must always be excluded, if possible, because of its tendency to produce perichondritis, chondritis and necrosis. Lupus is prone to produce fibrosis, with or without infiltration, but almost always with very little if any edema. Laryngitis, complicating influenza or pneumonia, with perichondritis as the most noticeable laryngeal feature, may be due to any one of the pus producing organisms, particularly the pneumococcus. Actinomycosis produces laryngeal changes which may easily be confused with tuberculosis. Microscopic examination of a small piece of tissue which has been removed from the larynx will usually reveal the ray fungus.

Permit me to state, then, that while an early diagnosis does not always mean a complete recovery, we know that from 70 to 80 per cent of our cases of pulmonary tuberculosis are curable if the diagnosis can be made very early and proper treatment begun.

Brown[36] says that laryngeal tuberculosis "occurs in about 25 per cent or more of adults with pulmonary tuberculosis, slightly more in men than in women, and next to tuberculous enteritis and colitis is the most frequent complication of pulmonary tuberculosis, due most likely to direct infection of the part with the sputum. Even early cases—cases in the incipient or minimal stage—are not spared (12 per cent), but as the pulmonary disease progresses the laryngeal complication be-

comes more frequent (moderately advanced, 25 per cent; far advanced, 45 per cent). The significance, then, of a complication so frequently seen among patients with pulmonary tuberculosis cannot be exaggerated." Dworetzky[17] says, "The prognosis of laryngeal tuberculosis, although not easily determined, depends on many influencing factors: the pulmonary lesion, general condition of the patient, site and extent of the laryngeal lesion, early diagnosis, character of treatment, etc. To the author, however, it seems that one of the major influencing factors is the character of the lesion itself."

Thompson[3] says, in speaking of laryngeal tuberculosis, that progress is ready to hand in the making of an earlier diagnosis of local infection. A laryngeal complication serves only to lessen a patient's chances of recovery and often prolongs the time required for an ultimate arrest or cure of the disease. Freudenthal[15] believes, "The prognosis of an established tuberculosis of the upper air passages is better than it used to be years ago." Greif[37] expresses the same opinion. Thomson[38] says, "Tuberculosis is one of the most common and most deadly scourges of humanity. There is no other which slays so many of our people in the very prime of their career, for it causes one-third of the total mortality during the chief working years of life. It kills 53,000 individuals annually in the small population of England and Wales. It is the greatest cause of disablement in adult life. It leads to more loss to the family and to the nation than any other single disease. It is one of the saddest afflictions." Coakley[39] says that laryngeal tuberculosis is most frequent between the ages of 20 and 45. and this corresponds with our experience. It strikes the hardest at the age when the patient should be of most use to the community in which he lives.

Freudenthal[15] advocates that all patients suffering from pulmonary tuberculosis should be advised to undergo a laryngologic examination, not only when the disease is diagnosed or upon their entering a sanatorium, but also at regular intervals, irrespective of their complaints. Mullin[40] has urged such an examination. Certainly it behooves all of us as laryngologists to be on the alert for the early manifestations if we are to succeed in curing tuberculosis.

PHYSICIANS' BUILDING.

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VI.

FUNCTIONAL HEARING TESTS IN NORMAL CASES.

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The problem of determining the acuity of hearing has of late years been approached from many different fields of science. A glance at the recent journals of physics or electrical engineering shows that the solution of the problem is of vital importance to investigators in these fields. As a rule, when acuity determinations are to be made, observers are selected who are of mature age and who feel that their hearing has not been impaired by accident or other pathologic condition, a selection which appears rather haphazard from the clinical point of view. The examinations are usually with but one type of apparatus and are impossible of interpretation to the clinician because no correlation is made between these laboratory tests and those of standardized clinical procedure. On the other hand, the reports of clinical examinations are chiefly concerned with cases where pathology is either known or strongly suspected.

The purpose of this study is to determine if these socalled "normal" cases are "clinically normal." With this in mind, a series of the usual clinical tests were made on five groups of university students from the School of Music, Department of Speech and the Training School for Nurses, comprising a total of one hundred sixty-four persons, of whom eighteen were men and one hundred forty-six were women. These were selected because of their mature physical and mental development, and because it was thought that they were not sufficiently advanced in age for the physiologic and pathologic changes of later adult life to have become apparent, and would represent a group similar to those usually selected for laboratory tests. This gave a more or less homogeneous group of observers who were in all probability of more than average intelligence. The tests were made a part of the regular class work

and consequently all evinced a desire to make the best possible records. It was expected that some would have auditory defects of which they were cognizant, but it was thought that these observers would represent what might be found in any ordinary group of mature young people.

The following age distribution chart shows that the majority (89 per cent) were between 18 and 28 years of age:

Age	No.	Age	No.
17	1	29	1
18	17	30	3
19	34	31	1
20	22	32	1
21	27	33	2
22	18	37	1
23	13	41	1
24	4	43	1
25	7	44	1
26	5	46	1
28	2	50	1

Age Distribution Chart.

The number of tests given varied somewhat with the different groups, depending upon the time allotment. The Rinne test, the Weber, bone conduction (Schwabach), spoken and whispered voice, the upper limit for air and bone conduction with the monochord, and the audiometer tests were made on all observers. These were supplemented in certain groups with the lower limit determinations by means of the large weighted Bezold fork (16 d. v.), by the complete octave series (50 d. v., c, c^1, c^2, c^3, c^4, c^5), by a special fork of 3,100 d. v., and by upper limit determinations with the Galton whistle and the Koenig cylinders. Since the audiometer covers in greater detail and accuracy a wider range than that of the Bezold forks, in the last series of forty-eight cases the tuning forks were used only to verify the results secured from the audiometer test. In addition, a short detailed history was taken, and when possible the tympanic membranes were examined by one of the clinical assistants. The time allotted to each observer varied from thirty minutes to one hour, depending on the number of tests given.

All tests except those for spoken and whispered voice were conducted in a small room adjacent to the Ear, Nose and Throat Clinic in the University Hospital. This room was by no means soundproof. Frequent disturbances made repetition necessary. The conditions, however, approximated those of the usual clinical procedure.

The Weber Test.—This test was made in the usual method. The weighted c^2 Bezold fork was held in the midline of the head and the observer asked to state whether the sound was localized to either side or if it was in the midline.

Results.—Eight observers gave a lateralization to the right and sixteen to the left. The rest were negative. Analysis of these cases reveals the following facts:

V. G. localized to the right, reports that both ears discharged twelve years ago, but notices no deafness at the present time. The audiometer shows a loss of acuity for low tones in each ear, and the right is distinctly poorer for tones above 1,800 d. v. Localization is toward the poorer ear.

F. K. localized to the left which is shown by the audiometer to be distinctly less acute. Has no history of ear trouble except a "feeling of fullness" with colds.

M. C. localized to the right, "the poorer ear." although all tests except the audiometer and tuning forks show the ears to be about the same. History of "abscess and running ears" six years ago.

A. G. localized to the right. Same results for all tests except the upper limit for the monochord is slightly lower in the left.

L. H. localized to the left. Indefinite history of ear trouble one year ago. Tests show the right ear slightly poorer.

P. S. localized to the right. Reports tinnitus in this ear. Is a member of the University Rifle Team. Tinnitus more noticeable after rifle practice. Holds the rifle to the right shoulder. Audiometer shows the right ear less acute for tones near 3,000 d. v.

R. E. localized to the left. Bone conduction, whisper and audiometer tests show the left to be less acute. No history of ear trouble except tinnitus at rare intervals.

A. I. localized to the left slightly. Is a concert singer. No evidence from tests as to the significance of the lateralization.

A. B. localized to the left. Reports earache and discharge as a child. Left ear less acute for tones between 1,200 d. v. and 4,000 d. v.

J. W. localized to the left. Believes her hearing rather better than the average. Has had considerable earache in the past. Bone conduction: right, —6 sec., left, —3 sec. Acuity tests show both ears to be the same. Lateralization toward the side having the better bone conduction.

P. S. is very much similar to the last case. B. C.: right, —7 sec., left, —5 sec. Lateralization is toward the left.

J. S. localized to the left. Is fifty years of age, the oldest observer in the series. Acuity tests better for the right ear.

O. M. localized to the left. History of acute ear trouble with influenza during the past year. Bone conduction: right, —2 sec., left, —1 sec. Acuity tests were equal. Localization toward the ear having better bone conduction.

L. T. E. localized to the left. Ears discharged for two months during the past year. Right tympanic membrane shows slight reddening while the left is normal in appearance. Left is less acute.

E. M. localized to the left. While there is no history of ear infection, both membranes show calcareous deposits, the left is more thickened, is less acute, but has the better bone conduction.

H. S. localized to the left. The Rinne is negative in this ear. Hears spoken voice at two feet. Ear condition diagnosed in this clinic as hyperplastic otitis media.

L. N. localized to the left. Acuity slightly decreased in both ears. Bone conduction: right, —4 sec., left, —2 sec. Ear condition was later diagnosed in this clinic as mild hyperplastic otitis media.

R. P. localized to the left. Acuity tests show the left to be less acute than the right for tones between 3,100 d. v. and 6,300 d. v.

A. D. localized to the left. No reason apparent for this lateralization, since both ears gave the same results for all tests.

E. C. localized to the right. Although all tests showed both ears practically the same, the observer stated that she thought the right ear slightly poorer. Both membranes showed slight retraction.

D. B. localized to the left. History of "abscess and running ears" at the age of six, but does not remember which ear was affected. Both membranes were gray and showed moderate retraction.

C. K. localized to the right. No history of ear trouble, but the left is less acute throughout the entire range.

M. B. localized slightly to the right. This tympanic membrane shows slight retraction, while the left is normal. Reports frequent sharp pains in the right ear recently.

Conclusions.—It appears from the analysis of these results that a history of ear trouble is a predisposing factor in determining whether the Weber shall be lateralized, but this is shown to be doubtful by the fact that thirty-four observers gave a history of ear trouble in the past which they distinctly remember, ranging in severity from a slight earache to a traumatic rupture of the tympanic membrane, but these do not lateralize the sound to the side known to have been affected.

The Weber test cannot in any sense be said to indicate the relative sensitivity of the two ears. In a large number of cases where pathology exists it is a valuable clinical indication, but a negative response to this test fails to indicate that no pathology exists.

The Rinne Test.—The weighed c^2 Bezold fork was used for this test also. Results were indicated simply as either positive or negative. In case of a positive result, no attempt was made to determine whether the result was "prolonged" or "shortened."

Results.—In the entire group, four ears gave the negative result. One, mentioned above as having hyperplastic otitis media, was negative. One had a history of traumatic rupture of the tympanic membrane when a leaf from a cornstalk penetrated the external meatus, an accident which happened twenty years prior to the time these tests were made, and a third gave a negative response in both ears, showing a loss of acuity in both ears, and, although not diagnosed in this clinic, was evidently pathologic.

Conclusions.—The positive Rinne is the usual response in all "normal" cases. In this series, the negative Rinne was always accompanied by ample evidence of pathology. Many showing evidence of pathology, however, gave the positive

response. The Rinne test as conducted, unless negative, gives little information as to the general condition of the ears.

Bone Conduction.—The handle of the lightly vibrating c^2 Bezold fork was held firmly against the temporal bone posterior to the auricle and on a level with the superior wall of the external auditory meatus, the experimenter being careful each time that in individual cases there should be the thinnest layer of musculature possible between the handle of the fork and the bone itself. As soon as the observer declared that he no longer heard the fork it was quickly transferred to a similar position on the head of the examiner, and the difference in the perception time noted. Results were determined from several trials, and if questionable an attempt was made to verify the result obtained by using the weighted c^1 fork in the same manner.

Results.—The results of the examination are illustrated in Figure 1. The interpretation of these results is a somewhat speculative matter since it involves a comparison. It must be assumed that the bone conduction of the examiner is either normal, increased or decreased by a certain definite amount. If it is not normal, clinical experience during the last five years has failed to reveal the fact. All the observers except five were of less advanced age than the examiner, and if age is a factor in determining bone conduction, that of the examiner should be less than the majority of the observers. On the other hand, the clinical assistants report that the tympanic membranes of the examiner show some retraction. In addition to this, the audiometer shows that he has some partial defects in the tonal range of each ear. The greatest difficulty, however, appears to be in getting the observer to concentrate his attention on the faint sound of the lightly vibrating tuning fork. It seems highly probable that the examiner may with constant practice give an increased reaction over untrained observers. It does not seem probable that such an advantage would permit him to hear the sound more than one or two seconds longer than the untrained observers, for at this stage the sound is quite audible to him.

The curve shows that nineteen observers had the same bone conduction as the examiner, while two hundred forty-one had a decrease of more than two seconds.

The four ears having increased bone conduction gave such a history that they may be considered pathologic. One shows a loss in acuity for all tones and had a negative Rinne: one had the tympanic membrane punctured by a blade of corn: one had the increase in one side only, gave a history of discharge from both ears during the past year but had a positive Rinne at the time these tests were made.

Of those having normal bone conduction, five observers gave this result in both ears, four had a decrease of one second in the opposite ear, three had —2 sec., one had —3 sec., and one had bone conduction increased in the opposite ear. Two having normal bone conduction had tympanic membranes which were normal in appearance and gave normal results in all tests. Two report earache in childhood and show slight defects in the range of tones covered by the audiometer. The fifth case has slightly retracted membranes and notices slight deafness when she catches cold. H. R., who had bone conduction in the left ear normal and in the right was decreased one second, gave one of the best audiometer records in the series and all other tests were normal. The other cases having normal bone conduction show slight depressions in the audiometer range and usually a history of some form of ear trouble in the past.

The curve also shows that a large number gave results indicating a decrease in bone conduction of from two to six seconds. Of these, sixty-nine (138 ears) have no recollection of ear trouble of any kind and all believe their hearing to be excellent.

Conclusion.—From a statistical viewpoint the results secured from the bone conduction test lie in a curve which closely approximates a curve of normal distribution, and as such will represent what will be found from the results of any standardized test on a large group of "normal" individuals. If decreased bone conduction is one of the clinical indications of nerve involvement, then it would appear that a large percentage of people of early adult life show this symptom. Its importance from the standpoint of prognosis as well as its reliability in group tests of this nature is a problem for extended study.

The Spoken Voice.—The tests for determining the hearing power for low conversational voice were conducted in the clinic room, which is approximately 55 ft. by 12 ft. by 12 ft. The room is by no means ideal for hearing tests, but was the only large room available. The Barany noise apparatus was used in every case to exclude the ear not being tested. Numbers from one to one hundred were used for test words, care being taken to exclude the sibilants. Many factors of error undoubtedly influenced our results. There were frequent distracting noises about the room. The Barany apparatus is in itself a distraction. The room with its smooth reflecting walls might be considered a speaking tube of large dimensions. The greatest errors were in the selection of the words and the variation of the intensity of the examiner's voice. These errors were minimized as far as possible, and the results are certainly on a par with similar tests under clinical conditions.

Results.—The results are illustrated in Figure 2. It will be seen that the curve in no sense approaches a curve of normal distribution and consequently is of little value from the statistical point of view. From the clinical side, the fact that all but seventeen ears were able to hear the voice at 45 ft., the distance which is considered normal for this room, indicates that the individuals of the groups would under usual conditions be classed as having normal hearing if other tests were not considered. It is scarcely necessary to state that those showing defective hearing for spoken voice also gave other evidence of pathology.

Whispered Voice.—The conditions under which the whisper test was conducted were the same as those of the previous test. It is, therefore, subject to the same general criticisms.

Results.—The results secured are illustrated in Figure 3. Comparison of the records for the spoken and whisper tests shows a much wider variation in the latter. This may be accounted for by the fact that the possibilities of sound reflection from the walls of the room were largely eliminated by the use of the faint whisper. There appears to be a distinct relation between the whisper test, the bone conduction test and also certain features in the audiometer records, points which will be brought up later in this report.

The Upper Limit. Results.—Figure 4 shows the upper limits secured by means of the monochord for air and bone conduction, for the Galton whistle and for the Koenig cylinders. The whistle and monochord are calibrated in double vibrations to the nearest thousand. The cylinders are in terms of single vibrations, but are plotted for comparison in double vibrations to the nearest thousand. The curves show a higher limit for the cylinders than for the other instruments, a fact which may be accounted for because of the greater intensity of sound or perhaps by errors of calibration. The curve for bone conduction with the monochord is slightly higher than that for air conduction with this instrument. Two hundred thirty ears gave the same result for both bone and air conduction, sixty-one ears had bone conduction 1,000 d. v. higher, twenty-seven had bone conduction 2,000 d. v. higher, in seven it was 3,000 d. v. higher, and in one ear it was 4,000 d. v. higher. In the previous work which has been done with this instrument, evidence has been presented for believing that more comprehensive results are obtained by the use of the monochord than with the other instruments.* While there was considerable variation in results for the same individual, those shown to be distinctly low with one instrument were likewise low for the others. The majority of the determinations with the Galton whistle were between 19,000 d. v. and 21,000 d. v., with the monochord the peaks of the curves lie between 17,000 d. v. and 20,000 d. v., while with the Koenig cylinders it is between 20,000 d. v. (40,960 v. s.) and 25,000 d. v. (49,152 v. s.).

The Lower Limit.—The weighted 16 d. v. Bezold fork was used for this test. Care was taken in energizing the fork to prevent the appearance of overtones. Unfortunately, perhaps, as will be shown later, this test was not given to the music students.

Results.—Of the eighty observers (160 ears) who were given this test, the sound was audible to all except five. The five to whom it was inaudible gave other evidence of existing pathology.

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*See "A study of the tonal ranges in lesions of the middle ear," Dean, L. W., and Bunch, C. C., Ann. of Otol., Rhi. and Laryng., 1922, p. 617.

The outstanding difficulty with the test was in the analysis of the perception. Twenty-nine described the sound as a distinct "tone." Others replied that it was of the nature of a "rumbling," "like an aeroplane," "a vibration," "fluttering," "just motion," "a buzz," etc. Had the music students taken the test, more significant responses might have been obtained. If, however, musical training is essential for accurate results in the test, it is of doubtful clinical value. The fact that in five ears it was inaudible seems to indicate that tactile sensations were not responsible for the responses given.

Conclusions.—It appears from the clinical standpoint that the 16 d. v. fork is distinctly audible to "normal" ears. The tonal quality of the perception is a psychologic problem.

Audiometer.*—In this test, the observer was seated with his back to the experimenter, the left ear being conveniently tested first, a procedure which was followed throughout unless the observer knew the right ear to be more acute.

Results.—The best thirty-eight ears (11 per cent) gave records falling within the space *A*, Fig. 5. These were selected as the best because they gave the highest curves and there was no evidence of defects in the tonal range. Only one gave a lateralized Weber, this being toward the better ear in a case where the opposite ear was not included in this group. The Rinne was positive in all cases. All heard the spoken voice at 45 ft. and the whisper at 15 ft. Four had normal bone conduction; in seven it was decreased 1 sec.; eight were decreased 2 sec.: four were decreased 3 sec.; six gave —4 sec.; three gave —5 sec.; three, —6 sec.; two, —7 sec.; two, —8 sec. Of the thirty-eight ears, twenty-one were negative for ear trouble. The others report an indefinite history of "earache in childhood." The records of five other ears falling in this space were not included in this group because they were unable to hear the whisper at 15 ft.

Those who were classified in a second group, thirty in number, gave curves falling within this area except for a small area which is indicated in the figure by *B*. Fig. 6 shows the

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*For a complete description of the audiometer and procedure of tests see "The Measurement of Acuity of Hearing," Bunch, C. C., Iowa Studies in Psychology, No. viii.

results for the whisper and bone conduction tests for this group of observers. Fifteen had a history of past ear trouble.

Conclusion.—If conclusions can be drawn from a comparison of these two groups, it appears that a decrease in sensitivity indicated by the space *B* is the smallest evident and distinct loss characteristic of a group. It is further characterized by a loss of acuity for whisper and a decrease in bone conduction.

Group III is characterized by a loss in sensitivity for tones in the area indicated by *C*, Fig. 5, comprising the records of forty-eight ears. While some of these observers were deficient for the area *B*, they also show a loss for tones between 1,000 d. v. and 2,800 d. v. Fig. 7 shows that the percentage having a decrease for whisper and for bone conduction is greater than in Group II. This is difficult to interpret if we accept the statement of Miller (Science of Musical Sounds, Miller, D. C.) that the range of whispered vowels lies well within that of the spoken vowels.

For clarity, a single curve (Fig. 8*) is given to illustrate the findings of the next group of observers, numbering ninety-seven. The area indicating the loss of sensitivity has increased, especially for tones betwen 2,500 d. v. and 4,000 d. v. Not one was able to hear these tones on intensity step 1 of the audiometer. There are many variations in the group from the curve shown, but the general contour of all is similar. All could hear the spoken voice at 45 ft. The results for whisper and bone conduction are illustrated in Fig. 9. Only one heard the whisper at 15 ft., and only one had normal bone conduction. The peak of the curve is at —5 sec., while in that for the preceding group it was at —3 sec. This seems to indicate a distinct correlation between acuity for whisper, decrease in bone conduction and acuity for tones between 2,500 d. v. and 4,000 d. v.

Forty-nine ears gave curves similar to that illustrated in Fig. 10. These have been classified in a group because the partial defect at 1,800 d. v. which was evident in the last group does not appear and the loss for tones near 3,100 d. v. has

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*In Figures 8 and 10, the lower boundary of the area covered by Group I is inserted in the dotted line for purposes of comparison.

become pronounced. Fig. 11 shows that the loss for whisper and bone conduction is more marked than in the last group. A history of ear trouble was secured in twenty-nine cases, the defect being of recent origin in several and one was undergoing treatment at the time the tests were made. Comparison of this curve with the lower boundary of the area covered by Group I shows that the variation for tones below 1,600 d. v. and above 4,000 d. v. is almost negligible. These are the tones usually indicated by the expression "low" and "high" tones.

Among the curves not already described are many showing variations from the types illustrated. To enter into detail with each of these would be unnecessarily tiresome. Fig. 12 shows the records of several of these cases. Variations may occur in any portion of the tonal range. Since all show marked losses in sensitivity they are in all probability pathologic and, as this study is chiefly concerned with nonpathologic cases, a discussion of these is beyond its scope. All these observers were aware of hearing defects and were either undergoing treatment at the time the tests were made or had had such treatment in the past.

GENERAL CONCLUSIONS.

The application of the functional tests of hearing to normal cases reveals the fact that auditory defects of various types are present in a majority of cases. The decrease in acuity most frequently found is for tones between 2,500 d. v. and 3,100 d. v. This defect expands in more advanced cases to include the tones from 1,000 d. v. to 4,000 d. v., often without apparent effect upon the upper limit of tonality. There is a distinct correlation between this defect, the loss of acuity for the whispered voice and a decrease in perception time by bone conduction. Seventy-three per cent of the cases tested had decreased bone conduction and in fifty-six per cent this decrease was more than three seconds, a condition which can scarcely be accounted for by inattention on the part of the observers.

The upper limit of audibility is well under 25,000 d. v. Determinations made with the monochord showed a higher correlation with the other clinical tests than did those secured by the Galton whistle and the Koenig cylinders.

A lateralized Weber was found in 7 per cent of the cases tested. They are accounted for by existing conditions of pathology or by assymmetric conductivity of the cranial bones.

These examinations were made in a region which is subject to rather abrupt climatic changes which are conducive to affections of the nose and throat. This fact, together with the above indicated symptoms, would apparently indicate an early appreciable involvement of the perception apparatus in a majority of cases of supposedly normal ears.
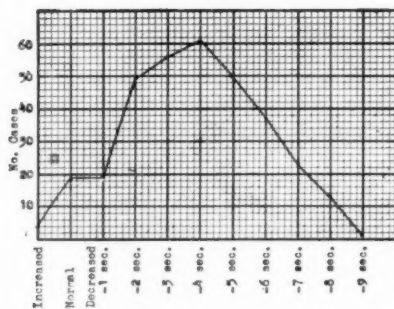
Fig. 1.
Results for Bone-Conduction.
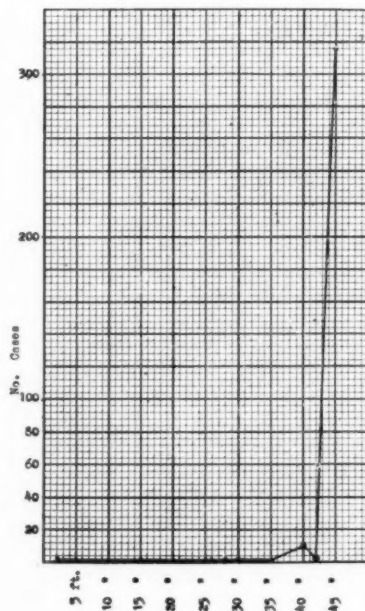
Fig. 2.
Distance for Voice.
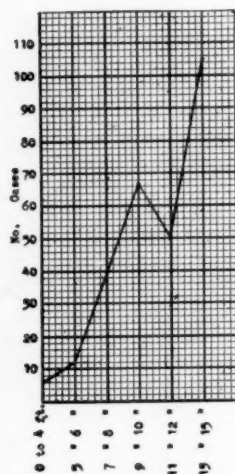
Fig. 3.
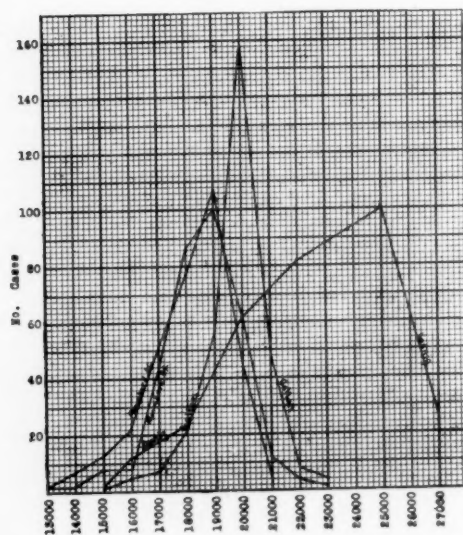
Fig. 4.
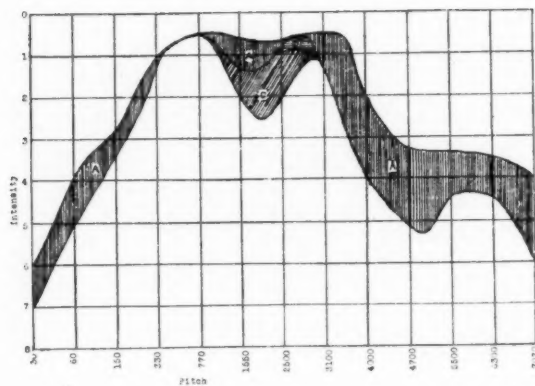
Fig. 5.
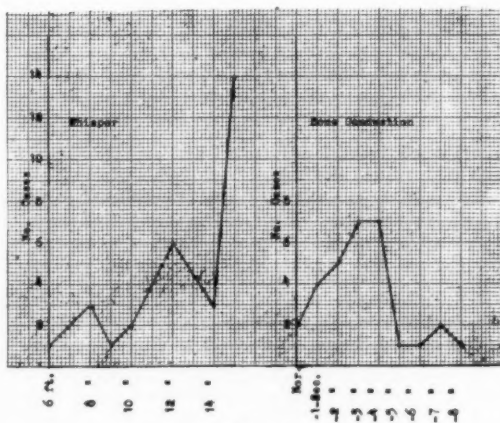
Fig. 6.
Bone-Conduction and Whisper of Group II.
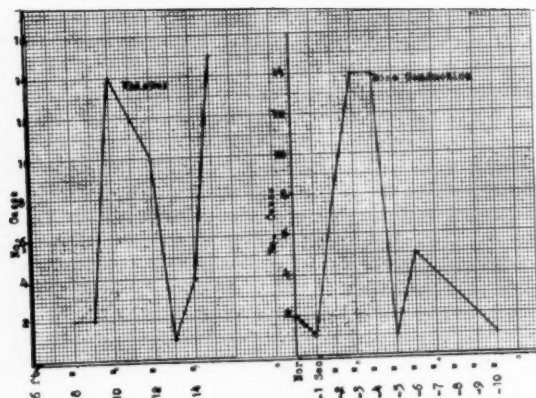
Fig. 7.
Bone-Conduction and Whisper of Group III.
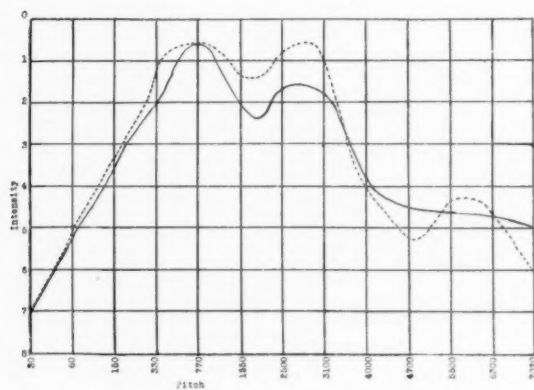
Fig. 8.
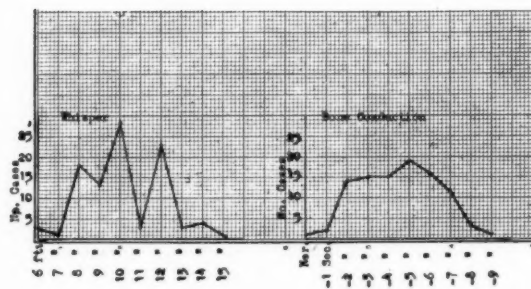
Fig. 9.
Bone-Conduction and Whisper of Group IV.
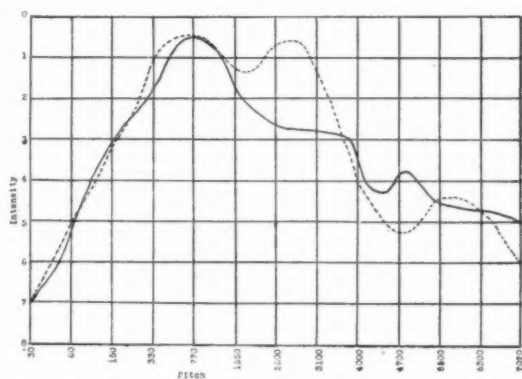
Fig. 10.
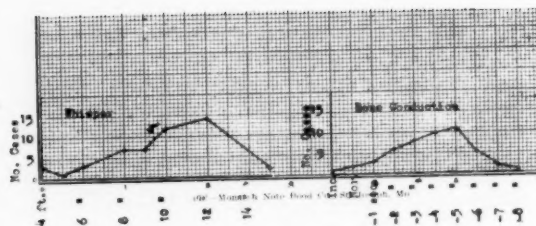
Fig. 11.
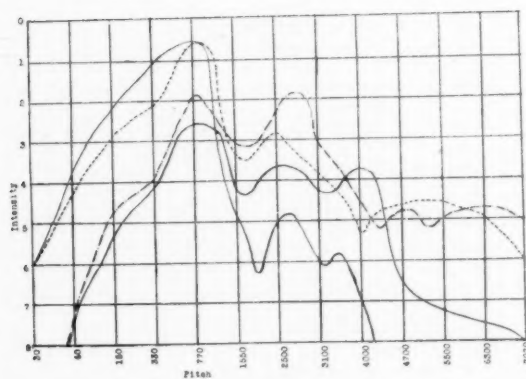
Fig. 12.

THE ELECTROCAUTERY IN THE TREATMENT OF LARYNGEAL TUBERCULOSIS.

By Joseph B. Greene, M. D.,

Asheville, N. C.

The electrocautery is not a new method of treatment for laryngeal tuberculosis, but the fact that it has not been as fully accepted by our profession as it deserves is my reason for presenting the subject to you at this time. Gruenwald, back in 1907, strongly advocated the application of the cautery for tuberculosis of the larynx, and in this country Wood, Fetterolf, Freudenthal, Levy and others have urged its more general use in this condition. The fact that such a multitude of drugs, as formalin, lactic acid, nitrate of silver, methylene blue, scarlet red, iodoform and more recently chaulmoogra oil have been recommended from time to time by various writers would indicate that none of them has proven entirely satisfactory. In my opinion, the cautery is destined to become the chief therapeutic measure at our disposal, for the reason that it fulfills two important requirements in that it is curative and also that it relieves pain. Then again it is suitable for almost any type of lesion. It accomplishes within a comparatively short time what drugs fail to accomplish after swabbing continued over a long period of time. St. Clair Thompson recently said in a paper before the American Laryngological Association (1919) : "I have given up lactic acid and practically all chemical caustics are found to be ineffective compared to the galvanocautery. It is the one treatment, not only for ease but for cure." The cautery stands somewhere between the ultraconservative form of treatment, where nothing is done save rest to the voice, with perhaps a cleansing spray, and that of Heryng and Krause, who advise such radical measures as removal of the tuberculous lesion by the curette or forceps. The cautery is not a radical measure, for the reaction is usually slight and delicate structures are not

sacrificed. No attempt is made to remove all the diseased tissue as is the case with malignant growths, but rather to stimulate the formation of new blood vessels with later development of scar tissue. Wood has well shown by a series of experiments that tubercles artificially produced in the skin of guinea pigs disappeared by the use of the cautery. A microscopic section showed that the favorable action extended well beyond the site of the cautery needle. These experiments so heightened his enthusiasm for this form of treatment that Wood has practically abandoned all other therapeutic measures in the way of topical applications. This method of treatment is usually not painful and spares the patient the exertion and fatigue incident to frequent visits to the doctor's office. As a rule, an interval of several weeks is required for the reaction to subside before a repetition of the cautery in the same locality is advisable, though a shorter time may elapse should an entirely different area be attacked. It is usually safer not to attempt too much at one sitting in order to avoid unpleasant reaction. I have a patient now under my care for tuberculosis of the middle ear whose epiglottis was treated with the electrocautery seven years ago by Fetterolf. He has now a perfectly healed stump of an epiglottis, with not the slightest evidence of a recurrence. I would like to say just a word about rest of voice as carried out by silence. There is hardly a question but that it proves a very useful measure in certain types and stages of the disease, but to practice it as routine to the exclusion of all other forms of treatment is, in my opinion, not doing the best possible for our patients. In the early stages of the disease, evidenced by general redness, situated other than the epiglottis, there is hardly a doubt that vocal rest, as far as it can be attained, is a very valuable measure. In fact, I am inclined to think it should be the sole reliance at this stage so far as local treatment is concerned. It has never seemed to me that old infiltrated areas associated with a good deal of scar tissue wherever situated are in the least favorably influenced by vocal rest. We must remember that perfect rest to the larynx is very difficult to obtain. Whispering, unless executed by the lips, does not take all the burden from the larynx, and coughing, as we know, places a great strain on the vocal apparatus. The use of the pad and pencil or the

sign language are far preferable to the use of the whispering voice. The great advantage of the electrocautery is the hopeful prospect of early healing of the lesion and a return to the use of the natural voice. This change to a moderate use of the voice often works wonders for the patient from a mental standpoint. In reference to contraindications, will say that in late stages of the disease, with extensive laryngeal involvement, particularly if the chest condition is bad, the use of the cautery is hardly indicated, unless it be to sear ulcerations for the relief of pain. In these cases injection of the internal laryngeal nerve with alcohol or a cocain spray may have to be resorted to. As before referred to, I am not in the habit of using the cautery in the early or socalled congestive type, particularly where there is general redness involving the vocal cords. In fact, the cautery is rarely applied to the cords unless there be a well localized infiltrated area which can be readily reached by the cautery. However, if there be a localized ulcer on the cord which shows no tendency to heal, the cautery may be necessary to prevent further destruction of tissue by the necrotic process. Prominent tuberculosis infiltrations often seen in the posterior commissure respond well to the cautery, with marked improvement in the voice. Lesions of the vocal bands do remarkably well under this form of treatment, though care must be taken not to cauterize too deeply, and not to attempt too much at one sitting. Large and swollen arytenoids shrink markedly after a few punctures with the cautery needle. The pain so often associated with this condition is often ameliorated. In fact, since I have adopted the cautery in my laryngologic work I less frequently resort to blocking the internal laryngeal nerve with alcohol. Tuberculosis of the epiglottis is, in my opinion, the most painful area of the larynx and the least benefited by injection of the internal laryngeal nerve. It is often astonishing how rapidly the pain disappears after a few punctures with the electrocautery needle. If there be ulcerations, these should receive superficial cauterization. I do not share the opinion of those who hold that the adoption of the cautery has entirely supplanted the surgical removal of the epiglottis. There are cases where this seemingly radical procedure is entirely justified, particularly when the ulceration is situated near the tip and

is causing pain unrelieved by any other measure. Then again, as massive infiltrations of this region disappear very slowly by cauterization, much time can be saved by removal of at least a portion of the epiglottis. The stump should then be treated by the cautery.

Technic.—In the use of the cautery I have largely followed the method of Wood, who has been a strong advocate of this form of treatment for a number of years. It is my custom to practice the indirect method, though I realize that skilled laryngologists, like Arrowsmith, Lynch, Dean and others prefer the direct method, using the suspension apparatus. It has seemed to me simpler and perhaps more comfortable to make the application by the indirect method, making it simply an office procedure. However, every well equipped sanatorium for the tuberculous should be equipped with a treatment room where patients in a weakened condition can be spared the exertion incident to a visit to the doctor's office. As to the anesthetic, will say that the pharynx and larynx are first sprayed with a 4 per cent cocain solution. The laryngeal applicator, well protected with cotton, is moistened with sterile water and then dipped into cocain flakes. The larynx is next thoroughly anesthetized, using at least three applications at intervals of several minutes. Inasmuch as every tuberculous larynx presents a different picture it is needless to say that it would be impossible to give a technic that would be suitable for every case. However, as a general proposition, would say that ulcerations require a superficial cauterization with a knife, and infiltrations, except within the larynx proper, require needle punctures. As a rule, it is better to be satisfied with not more than three or four punctures at one sitting. It is my custom to enjoin absolute silence for about a week after the use of the cautery, except where the epiglottis is being treated, returning gradually to the whispering voice. I frequently advise the use of cracked ice held in the mouth at frequent intervals for several days following the use of the cautery. It seems to lessen the reaction and add to the patient's comfort.

Although the title of this paper might proclaim a discussion of pharyngeal tuberculosis, I would like to say for ulcerations in this region there is no other form of treatment which will

compare with the electrocautery. This has no reference to the miliary form of the disease manifested by superficial grayish patches on the pharynx. This type of the disease often indicates a general dissemination with a lowered resistance, and is as hopeless here as when manifested elsewhere in the body.

In conclusion, I wish to say that no field of our special work is more deserving of untiring efforts than the treatment of laryngeal tuberculosis. If I have succeeded in arousing your interest in the treatment of a condition which promises so much for the patient's cure and comfort then I shall feel fully justified in having taken your time in the presentation of this subject.

305 HAYWOOD BUILDING.

VIII.

FUNDAMENTAL CONSIDERATIONS UNDERLYING ROENTGEN THERAPY OF TONSILS.

By Eugene R. Lewis, M. D.,

Los Angeles.

I desire above all things to make myself clearly understood at the outset of this discussion concerning one point, namely, that I realize that nothing in the way of argument can be sufficient to counterbalance the actual testimony of clinically proven case results. Whatever I may say in this discussion is actuated by a single-minded regard for the good of the patient, who places with confidence the high responsibility of his physical welfare into the hands of the physician. Without fear or favor, and as nearly as it can be accomplished, without prejudice, I desire to open the discussion as to the safety or danger, the efficacy or futility of the use of the X-ray in deal-in with the tonsillar conditions with which we are confronted. I desire also to state clearly that there is in my mind a definite conviction that no one is justified in undertaking any radical treatment of tonsils lightly and with full assurance that no remote undesirable results may follow.

In this connection let me quote Pacini: "In the child the lymphatic system is dominantly active, and since the tonsils may really be considered an integral part of the lymphatic system, we may expect hyperactivity in those organs in early age." Those of us who are in accord with the teachings of Bushnell with regard to the immunizing dose of tubercle infection sustained in childhood by repeated ingestion and aspiration of tubercle bacilli, can see in the tonsils a locus through which the circulating tissue of the infant derives its immunologic defense against usual pathogenic bacterial flora. It is not improbable that the physical texture and the chemical constitution of the tonsillar glands are purposely adapted to bacterial culture; for, whether purposeful or not, in children it is frequently a finding to observe subacute infections commonly present.

Assuming that the tonsil, by reason of its position, its histologic texture, and its chemical constituency marked by lymphatic preponderance, is charged with the duty of collecting ingested and aspirated organisms, and also breeds pathogenic organisms in a subacute fashion, it is possible to conjecture a threshold of maximum activity at which pathogenic organisms thrive to a point where focal or systemic infection is not clinically established, though the products of their elaboration are osmosed in the lymph and circulated and distributed throughout the body, raising generally the immunologic titer of the tissues against various infections. Or, to speak in terms of an established science, it seems probable that the tonsils are likely to contribute, during childhood, to the establishment of an immunity, an immunity acquired at so early a date as to be inseparable from the defense usually recognized as natural immunity.

No argument is necessary to establish the realization that the acquisition of immunity, as of many other desiderata, remains in the balance for a considerable period of time until summation of influences pro and con determines definitely the outcome favorably or unfavorably for the individual. For example, the successful titration of the tissues against tuberculosis during the formative period confers lasting immunity to that individual; interruption at a crucial stage by some intercurrent incident or disease may bring about the cessation of this immunization, with the result that tubercular liability may ensue for the rest of the individual's life.

Who can say with conviction that any radical attack upon tonsils, happening at a crucial juncture, may not interrupt what might go on to successful complete immunization? Even though the immediate effect of such procedure may be unquestionably desirable in handling some such transitory condition as otitis, rhinopharyngitis, colds, growing pains or acute chorea? I do not believe we should refrain from tonsillectomy on any such grounds, but we must nevertheless bear in mind that we may be interfering with some other important physiologic process when we do a tonsil adenoid operation.

In my opinion, the often alleged charge that the tonsil is "enlarged" is unworthy of serious scientific consideration as a reason for its removal. The very nature of this structure

and of its role in the complex function of establishing and
maintaining resistance and immunity to bacteriologic or other
exogenous insults, is such that fluctuation in size may be ex-
pected in the course of its normal physiologic activities. The
almost universal reference to "reduction in size of tonsils un-
der treatment" furnishes to me no argument or proof of bene-
ficial effect. One has only to consider the vast amount of tes-
timony concerning the widespread and vicious systemic effects
of toxic dissemination from strikingly small and shrunken ton-
sils to realize how little the size of the tonsil has to be with its
good or bad effects upon the individual. On the other hand,
how frequently one sees large discreet tonsils in the perfectly
healthy.

Another fallacy which it seems to me requires no elaborate
argument to point out refers to the beneficent effect of con-
verting to a mass of fibrous tissue incapable of cellular func-
tional activities this highly specialized cellular structure, whose
sole function depends upon the possession of a full complement
of special lymphatic cells, without which it could not function-
ate as a lymphatic organ. The same kind of reasoning would
argue the advantage of converting normal liver and kidneys
into fibrotic organs by some such agency as alcohol or lead.
Let me adduce a quotation from Witherbee's article of April,
1922, in this connection: "The fibrotic tissues remaining after
X-ray treatment and encapsulation of this abscess (of the
tonsil) points out the fact that we leave only that type of
tissue which nature utilizes in her defense against infection."
Witherbee does not seem to realize the fact that these fibrous
cells which are left are inert and that the only tonsil cells
"which nature utilizes in her defense against infection" are
the lymphoid cells which disappeared, not because nature re-
quired connective tissues for defense against infection, but
because the actual infection fighting cells fought a losing fight
and suffered destruction.

IS THE PRESENT METHOD OF X-RAY TONSIL THERAPY DANGEROUS?

The writer has had no experience with the use of the X-ray
in treating tonsils. During the past two years much has been
written advocating the removal of tonsils by the X-ray; argu-
ments in favor of this procedure include: applicability to hemo-

philiac, status lymphaticus, diabetic patients; greater safety to all patients; avoidance of operative incapacitation, avoidance of operative and postoperative discomfort to the patient, conservation of faucial tissues, safeguarding against disturbance of pharyngeal function. In the writer's opinion, some of these arguments are specious, misleading and dangerous, for the reason that they make a compelling appeal not only to the patient but to the general practitioner, whose knowledge of the physiology of the important structures involved is not sufficiently intimate to protect him against misconception.

The prime proposition is that the X-ray is capable of accomplishing selective destruction of lymphoid tissue; the tonsil is composed of lymphoid tissue and septa of connective tissue. By subjecting it to X-rays the lymphoid cells are easily, painlessly and safely put out of existence, leaving the inert stroma intact. With respect to the prime proposition there are only two alternatives—either the X-ray is capable of selective destruction of the lymphoid tissue or it is not. In the latter event, its application to tonsils would be obviously useless, although it might produce no deleterious results. With this brief consideration of the second alternative, we can devote our attention to a consideration of the first—the really dangerous one.

Granting that the X-ray can be applied in such a manner and in sufficient dosage to accomplish selective destruction of the lymphoid tissue of the tonsil, let us see what must happen in the process of accomplishing this. The tonsil is one of a group of lymph nodes which have certain definite relations with the great lymphatics leading from the upper respiratory tract. These lymphatics drain the areas of pharyngeal, oral and nasal mucosa, probably of all body areas the most exposed to attack by infectious organisms. Toxic and infectious material, whose portal of entrance is in these especially exposed areas, is designed by nature to encounter this group of lymph nodes immediately upon its encountering the lymphatic system. Without entering upon a detail discussion of the protective mechanism and the manner of its operation, suffice it to say that were no provision made for the interposition of obstacles to the progress of the noxious material which has once gained access to the lymph stream, it would reach the

heart and be broadcasted by the circulation. The anatomic
obstacles to such immediate dissemination of this noxious ma-
terial are the lymphatics. When a normal tonsil is attacked
by virulent infection it reacts in an attempt on the part of
body cells to put to death invading microorganisms. It often
transpires that the invaders are sufficiently virulent to over-
come the resistance of the tonsil cells and gain deeper foot-
hold. In this event the retrotonsillar lymphatics come into
action against those invaders which have succeeded in passing
the tonsil. The invaders meet successively a second, third and
fourth line of defense—along the lymphatic avenues leading,
respectively, toward the mediastinum, tympanum, meninges
and the cervical triangles. In relatively rare instances infec-
tious invasions of supreme virulence accomplish extensive dam-
age and destruction to these successive lines of defense and
gain access to remote areas of the body, where they set up
such pathologic processes as mediastinitis, lung abscess, otitis
media, mastoiditis, cervical adenitis, endocarditis, endarteritis,
phlebitis, synovitis, osteomyelitis, myositis or such general
diseases as typhoid, poliomyelitis, encephalitis, etc.

Even granting that it has ceased to be an asset and has
become a liability, there is only one justification for proposing
to remove a diseased tonsil. That justification lies in the exist-
ence of the second, third and fourth lines of defense in the
lymph nodes backing up the tonsil. These nodes take the place
of the diseased tonsil in the defense activities of this area.

The tonsil differs radically in its economic relation to the
body from certain other organs. For example, should one kid-
ney become so changed by disease as to be converted from a
functional asset to a pathologic liability, it may be justifiable
to propose its removal, realizing fully that the proposed sur-
gery will deprive the body of half the organs which alone are
capable of subserving the specific function of the kidney.
Should both kidneys be the seat of such disease, it would be
unthinkable to propose their removal, and some other means of
meeting the situation has to be considered. In the case of the
tonsils, removal of both tonsils can be considered when they
have been converted from functional assets to pathologic lia-
bilities, for the reason that there exist other similar function-
ating lymph nodes, immediately behind and backing up the

tonsils, which are capable of compensating functionally for the tonsils in case of their loss by surgery or disease. Were it not for the existence intact of this second, third and fourth line of defense back of the tonsils, tonsillectomy would be unjustifiable.

If, with the foregoing statements in mind, we consider what is proposed when one undertakes to accomplish selective destruction of the lymphoid tissues of the tonsil by X-ray, the rejection of such a proposition seems unavoidable. The same ray which is to accomplish this selective destruction of lymphoid tissue of the tonsil traverses first the lymphoid tissues of the lines of defense backing up the tonsil—the lymph nodes. Whatever destruction of lymphoid tissue is accomplished in the tonsil is preceded by destruction of lymphoid tissues in the nodes traversed by the rays on their way to the tonsil. The military parallel of this procedure would involve blowing up the fourth, third and second line trenches first, in an attempt to abandon an outpost which has proven untenable.

This discussion can include only a mention of the thyroid, thymus, the pituitary and other important anatomic structures exposed to the rays. The results of the raying of these structures are worthy of most careful consideration in this connection.

That the X-ray is capable of destroying highly specialized glandular cells is attested by many experiences. The effect upon the thyroid and thymus cells are apparent from the results of X-ray exposures of these structures in cases of hyperthyroidism and hyperthymism. I have encountered in a brief glance at some of the recent records the report of one case of myxedema developing two years after the last X-ray exposure for hyperthyroidism; another report by Means and Holmes of Boston, of four patients developing myxedema following X-ray treatment of hyperthyroidism in from four months to five years following the last X-ray exposure.

We find that Witherbee states in the American Journal of Roentgenology, August, 1922: "As in the treatment of tonsils each side is exposed for four minutes, thus giving crossfire treatment to the gland, at the same time including the tonsils and adenoids." After describing the details of the "factors used": 7 inch spark gap, 5 ma., 10 inch distant, 4 minute time,

filtered through 3 mm. aluminum, given at two weeks' intervals, number of exposures depends on the progress of the case. Average cases require six to eight treatments. Careful observation and examination of the throat during the treatments are as essential as are the factors of technic." The nose and throat men of the greatest experience find themselves at a loss to know how to construe findings in examination of the throat and utterly unable to state that a certain tonsil is or is not diseased. How the X-ray man attains his ability to construe his findings during the observation and examination of these throats is a matter of interest and importance.

To quote Witherbee again, after describing cases which have been exposed to as many as forty treatments in the area of these glands, he says: "This has been done without indication of any signs or symptoms of impairment of function or normal thyroid, parathyroid, pituitary and parotid gland." The inability on the part of an observer to detect evidence of impairment of function of these little understood organs of tremendous importance is not sufficient to establish the conviction that they may not, nevertheless, be suffering as a result of their exposure.

It seems almost too much to require of the X-ray that it act as a destructive agency on one occasion and that it produce no effect upon the same cells upon another occasion. I feel a profound fear of the effects of such a powerful and little understood force. This fear is not baseless, as I have followed the gradual amputation of finger after finger and the ultimate death from epitheliomatosis of three roentgenologists of my acquaintance. I have witnessed the distressing results of unintended burns of the skin and subjacent tissues. I have no definite knowledge concerning all the pertinent facts, but I believe the frequently observed childlessness of the roentgenologist is not without relation to their exposure to the X-ray.

The employment of such a powerful agency as the X-ray under the direction of those unfamiliar with the physiologic and pathologic fundamentals of the field and neighborhood in which they are working is distinctly dangerous.

1920 ORANGE ST.

IX.

INTRASEPTAL IMPLANTATION IN ATROPHIC RHINITIS.

By Harry L. Pollock, M. D.,

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Atrophic rhinitis, associated with ozena, probably the earliest nasal disease ever described, has been the bane of rhinologists for many, many years. Various therapeutic agents have been utilized in years gone by to combat this condition, but up to the present time not one measure has been advanced that will satisfactorily ameliorate this disease. It is not the purpose of this paper to delve into the symptomology or pathology of this malady except in so far as it is concerned with the new method of treatment as employed by the writer.

Before taking up the specific subject of the treatment, it will be necessary to go into some detail concerning the etiology. In the April, 1911, number of the London Journal of Laryngology, Rhinology and Otology, J. S. Fraser and F. Esmond Reynolds gave a complete description of practically all the various theories advanced to that time, as to the cause of atrophic rhinitis. Each advocate had his followers and, as is usual in these diseases of unknown origin, each claimed an advantage as to his particular theory over all others. Grünwald, who claimed that the disease was secondary to a sinus infection, probably has the greatest number of adherents because in practically every case of advanced atrophic rhinitis we find a chronic suppurative ethmoiditis. In a smaller number of cases we find the other sinuses are also affected. The maxillary antrum, sphenoid and frontal sinuses are infected in the order named in the large number of cases which have come under my observation.

It is needless to state at this point that before expecting any improvement of symptoms, attention must first be paid to the various suppurative sinus conditions. In describing my procedure for the attempted cure of atrophic rhinitis, we must take it for granted that all the affected sinuses have received

proper attention, viz., aeration and drainage. Knowing full
well that we are dealing with an atrophy, both of the turbi-
nates and the mucoperichondrium of the septum, our watch-
word must be preservation of as much normal tissue in the
nose as is possible by the preliminary attention to the nasal
accessory sinuses. The second theory to receive our attention
is that of Zaufal and Hauptmann, which brings forth the idea
of abnormal patulency of the facial bones, which makes the air
spaces in the nasal chamber much larger than those found in
normal individuals and which they believe is the causative fac-
tor in atrophic conditions of the nose. The ozena or stench
is no doubt produced by the action of the saprophytic organ-
isms developing in a fertile, moist, warm media in the nasal
chamber. The fact that we almost invariably find the Abel
bacillus or the Perez diplobacillus fœtidus does not necessarily
mean that they play an important part in the etiology of the
atrophic condition, but rather in the production of the decom-
position which gives rise to the exceedingly obnoxious odor.
By constantly removing the dried secretions, crusts, etc., and
keeping the nasal cavities clean by constant suction, we can
soon obtain relief from this odor and improve the condition
of the mucous membrane, but it will have no effect on the
atrophy or lessen the large space existing in the nasal cavity.
Taking this view of the causation of the crusts and odor, we
theoretically could cure the disease very easily by preventing
the discharge and crust formation. As just stated, we are able
to reduce the discharge by paying proper attention to the
sinuses, especially to the ethmoid labyrinth, which is most
always affected. Therefore, I recommend as thorough an
ethmoid exenteration as is possible, leaving the middle tur-
binate intact, doing the socalled Mosher operation, the plan
being to leave as much normal structure in the nasal chambers
as possible. The next consideration in the treatment consists
in reducing the space between the septum and the atrophic
turbinate. Zaufal, years ago, was the first to point out why
the crusts were formed. His explanation, which I believe is
accepted by almost everyone, is very simple—the immense
inrush of air dries up the watery secretion and leaves the solid
portion, which accumulates in the form of crusts. In addition
to this, my associate and I showed several years ago that the

fibrin content of the blood of patients affected with atrophic **rhinitis was double that found** in normal individuals. I feel that I now have a method by which the space can be lessened and the crust formation prevented, provided both patient and rhinologist persist undiscouraged. It is also important that this reduction of space must be permanent and not transitory if we expect permanent results.

There are three methods which have been employed in attempting to close the enlarged space:

1. By bringing the turbinate toward the septum.

2. By bringing the septum toward the lateral wall of the nose.

3. By bringing the whole lateral wall of the nose to the septum.

The first method has been but seldom attempted, and when tried the results obtained were very poor. Von Stein attempted to inject paraffin behind the inferior turbinate and gradually pushed it toward the median line. The method was not feasible and was soon dropped. The second procedure, viz., that of bringing the septum toward the lateral wall, has been tried with varying success. However, the method which I have employed has given uniformly good results. Dr. J. Wileminsky of Vienna, in an article entitled "Submucous Paraffin Injection" (Archiv. für Laryngologie und Rhinologie, 1906, page 458), was the first to give in detail a method of injecting paraffin into the septum submucously with the idea of bringing the mucoperichondrium out toward the lateral wall, thus lessening the space in atrophic rhinitis, and obtained some definite improvement in ozena cases. Dr. Harry Kahn of Chicago, in an article (Laryngoscope, 1911) entitled "Treatment of Ozena by the Submucous Injection of Paraffin Into the Nasal Septum," was the first American to call attention to this subject and from this method he claimed success. Both treatments consisted of using a paraffin needle, injecting the paraffin under the mucoperichondrium. After reading the article my associate and I conceived a method of introducing the paraffin which we thought would give better results. This consisted of making a small incision into the mucoperichondrium of the septum and elevating it as we do in performing the submucous resection of the septum, carrying the dissec-

tion down to the floor of the nose and at the same time attempt-
ing to elevate some of the mucoperichondrium of the floor.
This lower dissection is very important because of the greater
atrophy of the inferior turbinate and consequently the greatest
space is at the floor. After this careful dissection is made, and
here I would add that the elevation must be performed with
extreme care lest it tear the mucoperichondrium, thus defeat-
ing the result expected, sufficient moulded pieces of paraffin
are placed in this pocket, one by one, until the mucous mem-
brane is put on a slight tension. The edges of the primary
incision are then closed with a suture. The immediate results
are good, but the paraffin, being a foreign body, has a tend-
ency to slough out. I have had cases where the paraffin re-
mained in place for at least two years and then gradually
sloughed out. I have also had cases where the paraffin re-
mained in situ as long as I had the patient under observation,
the greatest length of time being four years, and, as far as I
know, it is undoubtedly where I placed it.

In all of these cases where I used the paraffin successfully,
the patients showed a most gratifying improvement, viz., ces-
sation of the crusting and a disappearance of the ozena, the
mucoid discharge being very slight. These implantations were
made unilaterally, and after a period of six months the oppo-
site side was treated similarly, provided the first side did not
slough.

Unfortunately, a very large percentage of cases sloughed
out. We then decided to try some other material which would
probably become organized and remain in place. Having had
several cases of cerebral hernia following decompression oper-
ations for various pathologic brain lesions, we found that the
best method of preventing a reformation of the hernia was by
employing fascia lata to cover the bone defect. The fascia
has been found to heal in nicely and become quite firm. I
therefore concluded to use the fascia for septal implants in
atrophic rhinitis, the fascia being very easily obtained from
the patient. I used the fascia in a number of cases, and found
that even after using the fascias from both thighs, on one side
of the septum, so much shrinkage occurred after a few months
that one could scarcely notice any bulging of the septum.

Therefore I discontinued this method on account of failure to secure the desired results.

I next attempted to use abdominal fascia, which was obtained by the general surgeon while doing a laparotomy. In nearly all of the cases in which I used the abdominal fascia, infection took place and the implant sloughed out, proving the impracticability of its use.

We then decided to try the Moosetig-Moorhof plug, which the general surgeons had employed so successfully in the treatment of osteomyelitis of the long bones. This plug consists of the following constituents: spermaceti, paraffin and iodoform. The method of introducing was also somewhat changed. The procedure was as follows: An incision was made through the mucoperichondrium and the cartilaginous septum and elevation made on the opposite side. For instance, if the right side was to be elevated, the incision was made from the left side of the septum through the cartilage. After the pocket was made, it was packed for about ten days with iodoform gauze so as to obtain a granulating cavity. The plug was then introduced into the newly formed cavity and the lips of the incision were sealed with collodion. Although we had never had an infection in any case in which we introduced the plug, sloughing occurred and the plug came out.

I then decided to use a homogeneous material, such as bone and cartilage of a septum from a known healthy individual previously operated upon. I felt confident that the bone and cartilage, being of homogeneous material, would remain in place and become a permanent part of the septum, provided that infection did not occur. It is now more than three years since I did the first intranasal bone cartilage implant, and the procedure has given uniformly excellent results.

The technic of the operation is the same as that used for the paraffin implant with some sligth modifications. The patient or patients requiring the submucous operation and who are to be the donors, are first tested to ascertain if they are free from any infectious disease, such as lues and tuberculosis. In my earlier cases I typed the blood of the patient from whom the septum was to be removed as well as that of the recipient, but found that it made no difference to which group they belonged and therefore have discontinued this practice. I

remove the septum under the most rigid aseptic precautions
and place it in dry, sterile gauze. Immediately upon the ter-
mination of the septum resection, the recipient is prepared for
the implant as before described. I do not permit the septal
cartilage or bone to be touched with anything but sterile in-
struments. After the mucoperochondrium has been elevated
and especially down to the floor, the piece or pieces of the
septum, including bone and cartilage, are introduced through
a Killian medium speculum, extreme caution being exercised
not to permit the implant to touch the mucous membrane of
the nose. In performing the submucous operation one should
attempt to obtain as large a piece or pieces of cartilage and
bone as possible and to use them as such, because it has been
the author's experience that the greater the number of pieces
one uses the more difficult it is to maintain the proper nutri-
tion, especially if the implants overlap. Therefore, the implants
should come in contact with as much of the mucous mem-
brane or septum as possible in order to assure proper nourish-
ment. It is also essential that the implants be placed as low
down toward the floor as possible, because it is here where the
greatest amount of atrophy occurs. I sometimes use a black
silk suture to close the incision, but more often seal its edges
with cotton dipped in collodion. I find that where there is
much atrophy it is necessary to use at least two septa, or other-
wise the space will not be sufficiently filled out.

In none of my cases did the pressure occasioned by using
two septa cause any necrosis of the mucoperichondrium and
consequent sloughing. In my first cases I made a grievous
mistake in doing both sides at once. We know that the septum
receives its nourishment from the mucoperichondrium, and
when I dissected both sides at one time all the nutrition was
lost and a necrosis of the septum resulted with a secondary
perforation at the site of the incisions. Since this unhappy
complication arose, I now wait for at least three months be-
fore attempting the second side. Owing to the fact that a few
of the patients objected to the use of material from other pa-
tients, and also that at times the patient did not desire to wait
until I had a septum or two to use, I have employed other ma-
terial for the transplant, such as costal cartilage from the pa-
tient's own ribs. The costal cartilage gives the same result

as the septal cartilage and bone.* In those cases in which we find a unilateral ozena or atrophic rhinitis in consequence of a marked septal deflection to the opposite side, the results have been most gratifying. This is very easily explained by the fact that the extra mucoperichondrium on the concave side, when dissected up and pushed out, is always large enough to come in contact with the lateral wall and leaves very little or no space in which crusts can form. After these cases have healed, the septum appears to have a deflection to both sides. We have no fear of using too much implant, for there is always a slight secondary shrinkage after the subsidence of the immediate postoperative swelling.

Naturally the first question which arises in our mind relative to this procedure is the following: Will the implant remain permanently or will it become absorbed? If not absorbed, what occurs? Clinically, I can state that after observing my cases for some three years, the implants are still as large as they were soon after the acute postoperative swelling subsided, and the clinical condition of the patients is excellent. Some of these patients still have a slight discharge, but practically all are free from odor and from the crust formations. I have no doubt that the same process takes place that we see in a bony or cartilaginous transplant in the correction of saddle noses when the transplants are brought into contact with periosteum or the bone itself. It obtains nutrition and continues to live. Carter of New York, in a series of cases of bony transplants which were brought into contact with the nasal bone, showed, by roentgenograms, that the bone continues to grow and remains as such for years after the implant is done.

I have just completed the third operation for implantation on one patient. The first was done on the right side one year ago. Having only one resected septum at the time, I made my mucoperichondrium incision fairly well back, expecting to implant farther forward at a subsequent time. Six months ago I transplanted the left side, and recently I implanted again on

*Since writing this paper, my associate, Dr. Joseph Beck, has employed a tibial osteoperiosteal implant in a case of marked atrophic rhinitis with most gratifying results. The implant healed in immediately and less reaction followed than usually occurs.

the right side, making my incision as far forward as possible
and dissecting backward as far as the previous incision. Just
posterior to it, I came upon firm fibrous bands through which
I could not dissect. These were probably connective tissue
fibers between implant, septum and mucoperichondrium, show-
ing that organization had taken place between implant and
surrounding tissues.

The third method consists in bringing the whole lateral wall
to the septum and attempting to form a synechia between the
inferior turbinate and septal wall. Max Halle, of Berlin, de-
scribed this method several years ago in an article in the
Berlin. klin. Wochenschrift (September, 1918) entitled "Fälle
zum Ozena Operations nach modifizierte Methods." His
technic is as follows: An incision is made in front of the at-
tachment of the middle turbinate, extending same in front of
the inferior turbinate, down through the mucous membrane
and periosteum. Another incision is made along the floor of
the nose, joining the previous incision. Then with a chisel he
cuts through the bone and forces this triangular piece toward
the septum. Just previous to this forcing the bone over, he
scarifies both inferior turbinate and septum, hoping to get a
synechia between these structures. He continues to pack the
opening for a long time so as to hold the bone in place. After
union has occurred, he removes the pack and allows the open-
ing to heal. He reports excellent results with this method.
Dr. A. Lautenschlager, in an article (Deutsche med. Wochen-
schrift, 1918), entitled "Operatives Verfahren bei vorgeschrit-
tenen Ozäna," modifies this method, or, as he claims, has sug-
gested a similar but new operation. He begins sublabially,
similar to a radical antrum operation, and after removing the
anterior wall of the antrum, chisels through and pushes back
the whole lateral wall of the nasal cavity toward the septum.
Dr. Schönstadt, of Berlin, in his paper (Berlin. klin. Wochen-
schrift, 1918) suggested another method in which he makes an
incision sublabially and then dissects up the mucous mem-
brane of the floor of the nose as far back as the choana. Into
this pocket he transplants a piece of tibia, thus hoping to lessen
the space in the inferior portion of the nose. All of these
operations are extensive and complicated and, I believe, incur
a great risk. I have not attempted either of these methods, as

I feel that I can obtain as good results by the method which I have described.

CONCLUSIONS.

1. The crusts are caused by the secretion arising from the suppurating ethmoiditis, losing its watery contents by the in-rush of too much air.   This is due to the large space in the inferior meatus caused by the atrophic inferior turbinate.

2. The stench is caused by saprophytic microorganisms, which find an excellent culture media in the moist, warm field.

3. The secretion can be lessened by exenterating the ethmoid labyrinth.

4. Both crust formation and fetid odor can be lessened or cured by reducing the space to its normal size.

5. This normal space may be produced by bulging out the septum toward the lateral wall of the nose by introducing bone and cartilage in the manner which I have suggested.

X.

THE CAUSES OF PERSISTENT OTORRHEA AFTER A SIMPLE MASTOIDECTOMY.

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A persistent otorrhea following a mastoid operation from the patient's point of view means an unsuccessful result. The surgeon feels, or should, that there may have been some fault in his operative technic or dressing. This does not mean that even in experienced hands we should expect 100 per cent dry middle ears, but with our present knowledge of mastoid surgery a dry middle ear within ten days should be the rule and not the exception. This is so far recognized in our large hospitals that when an operator fails to obtain a cessation of the discharge within these limits, certain recognized conditions are looked for to explain the results. While the different steps of the simple mastoid operation have been studied for many years and certain definite principles laid down, yet experience teaches that the detailed carrying out of those principles varies greatly with different operators. The ultimate success of mastoid surgery depends so much on the knowledge of the surgical technic and also on the immediate recognition of when the postoperative healing is going wrong that a general discussion may be of benefit to all. In adult cases, in the observation of the writer, the causes of a persistent otorrhea may be classified as follows:

First.—Perhaps no one cause is more frequently met with than lack of surgical judgment in the after care of a mastoid operation. No technic that the writer has observed is more efficient than that practiced at the Massachusetts Charitable Eye and Ear Infirmary for the last fifteen years. The external dressing is changed daily, if saturated or stiff from dried blood, and the wick changed in the external canal under strict

aseptic precautions. The mastoid packing is removed on the fourth to the sixth day, and a single wick is carried up to the aditus daily until the tympanic suppuration ceases. The bone cavity is then allowed to heal by organization of the blood and exudate without more drainage. Occasionally the lips of the external wound are opened with a probe to allow the escape of serum. If control of the aditus is not rigidly carried out until the tympanic suppuration ceases an otorrhea may continue indefinitely. On the other hand, long continued packing results in flabby and exuberant granulations, and a mixed infection occurs with part of the cavity epidermitized and the aditus wide open. The control of the aditus without allowing the blood clot and exudate to fill the exudated field and become organized until the tympanic suppuration has ceased seems to the writer to admit of no exceptions, and for this reason condemns the primary blood clot dressing as unsound in principle.

Second.—To early removal of the mastoid cortex before the infection has been limited by a leukocytic barrier. This was much more frequent before the days of the X-ray than at present. Unless the surgeon, however, has had a large operative experience his anxiety will lead him to operate while the bone infection is active and has not become limited by nature. Very rarely is it necessary to interfere surgically before a week from the time of an early incision of the membrana tympani. There are exceptions in measles and in some cases of influenza, but this is the rule. Very few patients die as the result of acute mastoiditis if an early incision of the membrana propria has been made without timely warning of danger. At times the process is so fulminating that the infection does not tend to wall off, but advances from one structure to another until a meningitis or general toxemia results in death. In these cases no operation would get outside the infected area. If all diseased bone is not removed the otorrhea continues. To get the best results, then, it is necessary to know when to operate in order to have the assistance of nature when the patient's resistance is best. We do not wish to encourage—far from it—delayed operating, but rather to point out that the experience of the surgeon as to when he shall advise the removal of the mastoid cortex is of

the first importance if there is to be a rapid cessation of the discharge.

Third.—Incomplete exenteration, especially of the deep layer of posterior canal cells. (It is taken for granted that we are discussing the procedure usually carried out in this country of opening the antrum in all cases.) If we remember that it is a straight line from the antrum to the tip cell this should rarely happen. Any mounding up between these points may mean that there are cells beneath that should be uncovered. Next to the posterior canal cells are one or more cells in the zygoma which are frequently overlooked.

Fourth.—To active surgery in the region of the aditus delaying the walling off of the middle ear and exposing the mastoid cavity to reinfection. While unhealthy granulations and necrotic bone should be removed here, as elsewhere, this should be done with a clear appreciation of what is necessary and what may be too radical surgery. In an acute process drainage is the important thing. Bone cells when not necrotic will take care of the infection if drained. This statement applies to a process that has become limited, for if this has not occurred our only safety lies in the radical removal of all cells, which is an anatomic impossibility. We never at any time remove all the infection, however radical the operator.

Fifth.—The active symptoms referred to the middle ear and mastoid and the necessity of operating upon a terminal process sometimes lead us to forget where the infection started. It is conceded that an infection virulent enough to cause an acute purulent middle ear and mastoid usually involves the paranasal sinuses and lymphoid tissue of the nasopharynx. The focal process may reinfect the middle ear and mastoid during the healing process and increase a discharge that was gradually diminishing. Unless this is recognized at the right time by the change in the character and amount of the discharge normal healing is impossible. This is shown by the case of a man seen two years ago at the Massachusetts Charitable Eye and Ear Infirmary on whom a double simple mastoid was done. The discharge was growing less when on the fourth day there was a profuse discharge from both middle ears and mastoid. He was admitted with a history of an acute exacerbation of a chronic tonsillitis. No other cause being

found, the tonsils were removed at once. The second day after the tonsillectomy the amount of discharge grew less, the character changed and both middle ears were dry on the tenth day. On the same service a mastoid failed to heal until an acute maxillary antrum was drained. These conditions are usually recognized in children but as often overlooked in adults.

Sixth.—Persistent otorrhea from arrested tissue repair may occur as the result of the poor resistance of the patient requiring general and local treatment. Stimulation locally may be carried out by the use of iodin-iodoform-balsam of Peru, etc., and necrotic bone tissue may need to be curetted. This should be done promptly if we are to succeed in walling off the middle ear in the required time. The patient's general condition should be fortified by tonics, forced feeding and fresh air. Every resource of the surgeon will be necessary to bring about normal healing, and the daily dressing should not be trusted to inexperienced hands. There are a certain number of cases where patients seem to have lost all power to react against certain types of infection. If the invading organism is active enough and the resistance of the host is lost, any type of infection may become chronic. Perhaps a staphylococcus infection in pure culture is more prone to degenerate into a persistent otorrhea. Good surgery done at the right time, with a proper appreciation of the problems involved, is usually successful in any type of infection, provided the diseased process has been limited by a leukocytic barrier.

Seventh.—Osteomalacia. This complication is fortunately rare, but when present is one of the most difficult to deal with.

Eighth.—General systemic conditions due to syphilis or tuberculosis. Lues in the eustachian tube or Rosenmueller's fossa may be the cause of a persistent otorrhea. Tuberculosis may cause unhealthy tissue repair or the formation of sequestra, especially in children.

The causes of a persistent otorrhea in children, on account of the greater danger of reinfection of the middle ear from the shortness of the eustachian tube and the greater activity of the lymphoid tissue, requires a separate consideration of their problems. Here, as in adults, an early free incision of the membrana tympanum is of the greatest importance.

Many, if not all, appreciate having this simple operation done at a time fairly near to the time of the infection. It is the writer's custom to have the adenoids removed in all cases at the time that a postaural is done. It is also important that a tonsil and adenoid operation should be performed at once whenever a patient is admitted with a history of a persistent or recurring otorrhea. Statistics from the writer's last hospital service teach their own lesson:

Nine cases that had been discharging from one to four months ceased to discharge in from five to fifteen days after a tonsil and adenoid operation.

Eleven cases, discharging from four to twelve months, ceased to discharge in from eight to fourteen days after a tonsillectomy and adenoid operation.

Seven cases, discharging from one to three years, ceased to discharge in from twelve to twenty-one days after tonsillectomy and adenoid operation.

Seven cases of acute and chronic suppuration showed marked improvement in the quantity of discharge.

Four cases showed no improvement at all.

While these statistics are very suggestive, we must remember that even in children the eustachian tube and middle ear may be reinfected from a pyogenic focus in the sinuses as well as in adults. In 32 children with otorrhea, where the infection was virulent enough to cause a postaural abscess, the pathologic condition in the antrum was demonstrated by radiogram in 24 cases. All these cases were under ten years of age and were examined consecutively as admitted to the hospital, whether they had symptoms referred to the nasopharynx or not. Where all the problems of mastoid surgery have been met intelligently, in the majority of cases we should have a dry middle ear on the fourth to the tenth day, and a healed process by two weeks. If this result is not obtained—that is, a dry middle ear within four to ten days—we should not procrastinate, but carefully go over the ground and determine where the fault lies without delay. Unless the discharge is obviously growing less within this time, the longer we put off correcting the cause the less our chance of obtaining a good result. A mixed infection results, granulations become flabby,

a healthy reaction is lost and a secondary operation is often necessary to stop the otorrhea.

The problem of persistent otorrhea is one, then, of continued infection or reinfection. This may obtain in the earliest years of life or in adults. Prenatal influences or abnormal anatomic conditions do not seem to the writer to be factors in the etiology or tendency to chronicity.

XI.

A NEW OPERATION FOR THE CURE OF OZENA.*

By Wolff Freudenthal, M. D.,

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As is well known, many writers in speaking of ozena still cling to the belief that it is caused by an infection. But, as you will remember, the mucous membrane of the nasal chamber has more power of resistance to infectious disease than the accessory sinuses; in other words, it is less frequently attacked by bacteria than the latter, early childhood and infancy perhaps excepted. For that reason alone an infection does not necessarily need to be the cause or the only cause in ozena. To say, as others do, that ozena begins in early childhood with an acute febrile process, an acute purulent rhinitis such as any child has at some time, followed by a chronic hypertrophic condition, etc., is a gratuitous supposition for which there is no proof. Nor do I believe that the bacillus of this or that investigator is the real cause of ozena. The bacterial infection occurs at a late stage of the disease; consequently the genesis of ozena has to be sought elsewhere, and the writer takes the liberty of drawing attention to the stand he took in a paper read before the International Medical Congress, held at Madrid (Spain) in 1903† and later amplified in an article on "Dry Catarrh."‡ The theories laid down in these articles will here be reviewed briefly in order to enable you to understand the surgical procedure proposed by the writer. The conclusion reached by me was that through our system of heating our houses and through other causes a desiccation of the mucous

membranes of the nose takes place, which results in a characteristic affection that is called rhinitis sicca. This may develop in any nasal cavity, whether or not hypertrophy be present. It may be an acute transitory condition or may become chronic. The details of this process are presented in the papers just mentioned.

If the factors producing a rhinitis sicca have been effective for a prolonged period, a conversion of hypertrophic into atrophic forms sooner or later takes place (provided a hypertrophy was present, which is not essential). For that reason, in the beginning of this transformation one may find in the rhinoscopic image as well as in microscopic sections the processes of hypertrophy and atrophy side by side. At a later date, the bony framework is affected by the same cirrhotic condition, and it is not surprising to discover the same changes as in the mucosa, changes which only gradually merge into the second stage, that of complete atrophy. We have seen—and so have others—cases of widespread atrophy in which the capacity of the nasal chambers was tremendously increased, but in which no odor was ever detected and no signs of ozena were ever found.

A still further and final development of this condition, however, is that into genuine ozena. Here the secretion has dried up, epithelial cells have disappeared, their ameboid action has ceased, and the formation of flat epithelial cells has followed. The latter cannot perform the work, and the deposited matter ordinarily removed by the epithelial cells stagnates. In short, we have here an atrophy of the mucosa and of the turbinals, just as in atrophic rhinitis; but in addition there are found masses of scabs and crusts producing the peculiar stench so characteristic of the disease. Therefore, it may be stated that ozena is nothing more than a rhinitis atrophicans plus an additional infection caused by some germ. Whether this microorganism is the bacillus of Loewenberg or Abel, the bacillus fetidus of Hajek or Pes-Gradenigo, the bacillus capsulatus of Friedlander, or the coccobacillus of Perez-Hofer has not yet been determined and is immaterial. One thing is generally accepted—that some type of bacterial infection is always present. But let me reiterate that none of these different germs will ever produce the clinical picture of ozena

unless the soil is prepared for it—that is, an atrophy of the
mucous membrane established beforehand. This, according
to my viewpoint, represents the pathogenesis of ozena, al-
though it is not in accord with the opinions of most other
writers, by whom many theories have been proposed. I would
mention particularly the "Herdtheorie," or theory of focal in-
fection, first advocated by Gruenwald of Munich and then by
Hajek. Others have claimed to cure ozena by operating on
the maxillary or other accessory sinuses. Still others, as Pol-
lock, first clear out the ethmoid cells, etc., and then attack the
ozena itself, and so on.

This brings us to the dominating view of the present time,
a view that is at the bottom of all surgical procedures pro-
posed of late. I am not referring here to the injection of
paraffin or of autogenous blood into the nasal mucosa but to
actual surgical procedures done with one idea in mind, viz., of
narrowing the abnormally wide nasal chambers. The principal
workers in this field have been Lautenschläger and Halle of
Berlin, and, more recently, Pollock of Chicago. Joseph C.
Beck worked years ago along these lines. To reduce the
abnormal patency of the nasal passages various methods
have been suggested. Halle and Lautenschläger accomplish
this by bringing the lateral wall nearer to the septum or, as
Halle still does, by forming synechiæ; or the septum is drawn
towards the external wall, as recommended by Pollock. In
May of this year Pollock read a paper before the American
Laryngological, Rhinological and Otological Society entitled
"Intraseptal Implantation in Atrophic Rhinitis," which has not
been published as yet, but which he kindly permitted me to read
afterwards. The Doctor implants new tissue into the septum
and claims universally good results. But whether these re-
sults will prove equally satisfactory in the future in his own
hands and in those of other rhinologists seems to me at present
immaterial. The main point at issue is the desirability of im-
planting new tissue into the nose, a procedure partly utilized
by Lautenschläger but in a different way.

The idea of implantation of new tissue also occurred to me
when I started experimenting along these lines. According
to the statement made by me above, the mucoperichondrium
of the nose is primarily and mainly affected, and the "periph-

eral ossification" (Lautenschläger) takes place at a later period. Considering this tissue to be chiefly affected, as I always have done, I have been building all my hopes on the possibility of its restoration. If one should succeed in reestablishing normal nutrition and circulation, the result would be a normal mucosa, and that would take care of everything else. This might be accomplished with or without a simultaneous procedure of narrowing the nasal chambers.

Acting upon these ideas, I began by transplanting autogenous, one piece epithelial grafts into the septum. I was induced to try such a method by the publication of several ophthalmologists on a similar plan of treatment of severe burns of the conjunctiva, etc. I would mention here especially an article by Dr. R. Denig on "The Early Surgical Treatment of Burns." Naturally, for the purposes I had in view, changes had to be made, and I finally adopted the following method:

First step: Removal of a portion of mucoperichondrium from the septum. The septum is anesthetized as for a submucous resection. Then a round piece of the mucosa, of the diameter of from $1\frac{1}{2}$ to $2\frac{1}{2}$ cm., is removed—that is, it is entirely excised. The size of this piece is governed by the size and accessibility of the septum, and the site of removal is close to the floor anteriorly or even including part of it. The nose is then tamponed in order to stop the bleeding.

Second step: Removal of a flap from the buccal side of the cheek. The inner surface of the cheek (for convenience sake, the side opposite to that of which the septal mucosa was removed) is anesthetized (novocain and adrenalin). Within a few minutes a graft somewhat larger than the nasal flap is excised. There is little bleeding connected with this step if you cut near the superficial layers, which alone are needed. As soon as you cut deeper into the tissues you are apt to get a hemorrhage. Furthermore, it is well to remember that a good deal of shrinking occurs in the graft, and for that reason it has to be larger than the area of excision in the nose.

Third step: Sewing up of buccal wound and trimming the flap. An assistant closes the buccal wound with a few sutures of catgut, while the operator at the same time trims the flap— i. e., he removes all capillaries and fat of the submucous tis-

sue. At this stage one has to bear in mind that only an epithelial layer is needed and not much more; and that the thinner the flap the quicker it can be adjusted. While trimming it, one has to dip it frequently into warm saline solution, which has to be filled up occasionally with warmer fluid in order to maintain a uniform temperature around 100° F. After the graft has been prepared carefully, and especially after all bleeding has been controlled and the cheek sewed up, we proceed to the

Fourth step: Transplanting the epithelial graft into the nose. With a thin forceps the graft is so adjusted as to cover the defect in the septal mucosa, the head of the patient being turned towards the other side. Naturally, it may have to be again trimmed a little in order to fill accurately the raw area. There must be no overlapping. Then a Killian speculum is carefully inserted, and by opening it the flap is gently pressed against the septum for a few minutes. The Killian speculum is now gently withdrawn, and the septum inspected finally to make sure that everything is correct. Finally, a small piece of cotton is introduced into the nose anteriorly, and the patient put to bed for three days. He is enjoined to keep quiet and not allowed to blow his nose.

I have operated in this way on a number of patients, and in perhaps the majority the results were satisfactory. This is not surprising, because before the operation there was a degenerated mucosa that never could accomplish the physiologic work required. With this new implant we obtain circulation of blood and nutrition, so that the entire mucous membrane of the septum can be, and actually is, benefited by it. But let me cite the histories of a few cases.

Case 1.—Miss C. G., aet. 29, gave the usual history of ozena of "long" standing. On December, 1920, the right side of the septum was operated upon under local anesthesia. She felt very uncomfortable the first three days, but there was no rise in temperature nor any reaction whatsoever. She came to my office on the fourth day, when I cleaned out the throat and the left side of the nose, and then the right nasal cavity (the one operated upon) by inserting a long speculum—of course without using any force. The graft adhered to the septum. From then on she commenced to feel more comfortable; she

called daily for two weeks, and then once or twice a week for
about six weeks. The formation of scabs had diminished ma-
terially, but when the nose was not washed out for several days
they formed again.

The patient is an intelligent woman, to whom my views had
been explained. After six months had elapsed she herself
asked to be operated upon on the other side. Consequently, on
June 22, 1921, a flap was transplanted into the left side of
the nose, with exactly the same course of events as before. The
improvement is very evident. Yet in winter she feels the ne-
cessity of calling on me about twice a month, since she no-
tices a good deal of dryness in the nose and throat, as well as
hoarseness to which she had always been subject. As soon as
warmer weather sets in and artificial heating ceases, she feels
easier, and I do not see her at all during the summer and fall.
On the whole, she is grateful for what has been done for her.
Objectively, there is still occasionally some slight formation of
crusts. The flaps have become an inseparable part of the mu-
cosa, not distinguished from the rest of it. The wounds of the
cheek healed completely within a week.

Case 2.—Arthur N., aet. 31, a school teacher, was operated
upon at three months' intervals, on both sides. He made an
uneventful recovery each time and now has very little difficulty
after a lapse of one year.

Case 3.—Miss L. M., age 19, had a typical empyema of the
antrum on the left side and at the same time a typical ozena
on the right side. She intended to get married within a short
time and asked to have both operations done simultaneously
and under general anesthesia. I was persuaded to do this, but
cannot advise such a procedure. First, the radical operation on
the antrum was done and then a graft transplanted. After all
was over, she vomited and we had difficulty in keeping the
flap in situ. I could keep her under observation for only about
two weeks, when she disappeared. The sinus had not healed
and the left side still showed some reaction.

Case 5.—Joseph F., aged 26, a carpenter by trade, had a
transplantation done on one side in May, 1922. This case
went wrong in more than one way. Whenever I started to
operate on his nose he drew up his face so that it was difficult
to obtain a good view of the septum. He remained at the hos-

pital only over night, and the next morning returned to his
home in an overcrowded tenement house district of Manhat-
tan. On the third day there was a slight rise in temperature
and his face was swollen. A stitch infection was the cause of
the trouble. This was the only instance in which a mild in-
fection was observed. I performed, by the way, a double mas-
toid at the same hospital right afterwards, and here, too, a
stitch infection was noticed (coincidence?). After removing
the stitches the fever and swelling subsided rapidly. The flap
was not entirely in place but had moved somewhat backward
and downward, so that a small part of the septum was bare—
i. e., without any mucoperichondrium. The result so far had
not been satisfactory. But my surprise was great when he re-
turned after about a year with a well healed wound. No de-
fect on the septum was visible at all, as this was covered
by an apparently normal mucosa.

Another patient, on whom I had operated bilaterally at two
sittings, showed, several weeks after the second operation, a
perforation of the septum. I am unable to say what the cause
of it was, since I did not notice anything of the kind during
the operation.

As this operative procedure for the cure of ozena is still in
the experimental stage, I am convinced that improvements in
the technic can and will be made. But one should always
bear in mind that the entire mucosa is defective and not merely
a portion of it. By my method I try to alter and improve the
circulation, etc. Lautenschläger, on the other hand, believes
that approximation of the lateral wall of the nose is the chief
feature in the treatment. This must be considered a funda-
mental error, as well as the theory that the abnormal patency
is the cause of ozena. The lateral wall is an integral part of
the nose but not the sole factor. To the same category belongs
the theory of the artificial formation of synechiæ between the
lateral wall and the septum. For several years past Lauten-
schläger has given this up entirely, while others still adhere
to it. The fact is, as Lennhoff of Berlin has said (Berliner
klin. Wochenschrift, p. 402, 1920), that formerly crusts and
scabs could be removed with great difficulty, but after the for-
mation of synechiæ they can be removed only by the physician.
The excision of a synechiæ afterwards is troublesome.

In conclusion, permit me to remark as follows: I have taken the liberty of bringing before this body of experienced rhinologists the outcome of my attempts to effect a cure in ozena. If any one of the Fellows should try this method, which should give excellent results in simple atrophic rhinitis, I would feel highly pleased. To me personally these experiments were of great interest, as they demonstrated how easily grafts of mucous membrane can be removed from the buccal cavity and how equally easy it is to transplant them into the nose.

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XII.

FURTHER EXPERIENCE IN THE USE OF TISSUE JUICES IN TONSILLECTOMY.*

By Joseph B. Greene, M. D.,

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In the removal of the faucial tonsils there are three chief considerations, aside from the anesthetic, which should ever be borne in mind:

1. The prevention of hemorrhage.

2. The lessening of trauma.

3. The avoidance of infection.

It is my opinion that infection is largely due to hemorrhage and trauma, and these causes can be greatly reduced by a technic which I shall describe later.

The clotting of blood is a very interesting and complex phenomenon, and we should make use in a larger way of this physiologic process in our surgical operations. The control of bleeding in tonsil surgery is a bit different from that which usually confronts the general surgeon, in that the gland is surrounded by delicate and important structures, and the region also is none too easy of access for the usual surgical measures. In throat surgery there is also the added hazard of inspiration of blood into the lower respiratory tract with the danger of a complicating pneumonia or lung abscess. These factors and others make it imperative that bleeding be prevented and controlled as accurately as possible.

The development of tonsil surgery has made wonderful strides within the memory of most of us. In the beginning the surgeon was quite satisfied with the removal of the presenting portion of the hypertrophied tonsil, the submerged or

inconspicuous looking gland not being disturbed. This was
in the days of the tonsillotome. In the next step of develop-
ment of tonsil surgery we find the operator quite satisfied,
and sometimes boastful, when he had succeeded in dissecting
the tonsil in its entirety, though the fossa show evidence of
marked trauma and shreds of muscle tissue be attached to the
gland. This marked the era of the early dissecting operation.
Thanks to the urging the late Hudson Makuen,[1] Kenyon[2]
and others, the technic of tonsil surgery has been so perfected
that we now have a feeling of disappointment, if not embar-
rassment, when injury has been done to adjacent structures.
This statement has no reference to the technic of tonsil re-
moval, whether by dissection or other methods.

It has occurred to me that at the present time more heed
might well be paid to the two important factors of hemorrhage
and trauma. Possibly the introduction of the suction appa-
ratus has been in a measure responsible for this seeming in-
difference to the loss of blood, in that the blood can by this
measure be so readily removed from the field of operation.
Although not wishing to enter into a discussion as to the ad-
visability of the use of the suction apparatus, for it has been
too generally accepted by our profession, yet I am of the
opinion that its use tends to encourage bleeding rather than
to favor clotting in the open vessels. While it is not my inten-
tion to discuss with you all the theories which have been of-
fered to explain the clotting of blood, yet it seems desirable
to recall with you some of the principles concerned in this
important physiologic process. In the first place, as you well
know, blood does not clot in the body during life, though the
important elements, thrombogen (prothrombin), calcium and
fibrinogen, are present. According to Morawitz[3], the element
thrombokinase is lacking in the circulating blood, though it
is present in shed blood being derived from the disintegrat-
ing white cells, particularly the blood platelets and the in-
jured tissue cells. It is thought that the thrombokinase of
shed blood, acting on the prothrombin in the presence of cal-
cium, converts it into thrombin, and this thrombin, acting on
fibrinogen, forms fibrin. Howell[4] terms this product, which
accelerates the clotting of blood and which is found in the
injured tissues and in the broken down white cells, "thrombo-

plastic substance." It has been noted that blood obtained without passing through injured tissue clots much more slowly than when brought into contact with wounded tissue or the tissue juices (tissue fibrinogen) of the body. The clotting of blood which concerns the surgeon is the normal clotting which takes place in accidental injuries to tissue or in surgical wounds. Brain surgeons, Cushing, Dowman[5] and others, have long made use of the application of muscle tissue to facilitate clotting in troublesome bleeding, and we have already at hand on removal of the tonsils these coagulent elements of the tissue juices at the very place where most needed. Tissues of the body vary greatly in the activity of their clotting elements, and it has been proved that the lungs, brain, kidney and skin contain this tissue fibrinogen in the greatest abundance. Recently Mills[6] has succeeded in making an extract of lung tissue which is more powerful than that obtained from the brain and other tissues of the body. It possesses such strong clotting qualities that 1 cc. injected into the vein of a rabbit caused death from general thrombosis in less than a minute. This same extract when mixed with blood plasma in proper proportions caused clotting within ten seconds. Tissue extracts which are on the market under various names, as thromboplastin (Squibb), hemolytic serum (Mulford), henagulin (Lilly), are obtained from calves' brains, while coagulin ciba (Fonio) and coagulose (Parke, Davis & Co.) are prepared from blood platelets. Although my experience with these preparations has been too limited for me to express an opinion as to their value, yet from a theoretic standpoint they might be at times useful.

Due to the kindness of Dr. C. A. Mills of the Biochemic Laboratory of the University of Cincinnati, I am able to give the activity of the clotting elements of the tonsils. The tonsils were taken from an adult, though Mills says children's tonsil tissue possesses practically the same degree of activity as does that of an adult. He says further in his report: "The yield (extract) was 0.9 per cent of the weight of fresh tissue, or about one-third the yield from lung tissue, which is the most active tissue in the body in this respect. This should rate the clotting activity of the tonsillar tissue as about equal to that of the kidney, testes and brain. I think you will find that it

is the injured tonsil tissue itself that aids in the control of hemorrhage." The result of the test follows:

1 cc. citrated horse blood + 0.35 cc. 1% $CaCl_2$ clotted in 13 minutes.

1 cc. citrated horse blood + 1.0 cc. tonsil extract + 0.35 cc. 1% $CaCl_2$ clotted in 55 sec.

1 cc. citrated horse blood + 0.5 cc. tonsil extract + 0.35 cc. 1% $CaCl_2$ clotted in 1 min. 10 sec.

1 cc. citrated horse blood + 0.3 cc. tonsil extract + 0.35 cc. 1% $CaCl_2$ clotted in 1 min. 20 sec.

1 cc. citrated horse blood + 0.2 cc. tonsil extract + 0.35 cc. 1% $CaCl_2$ clotted in 1 min. 35 sec.

1 cc. citrated horse blood + 0.1 cc. tonsil extract + 0.35 cc. 1% $CaCl_2$ clotted in 2 min. 0 sec.

I am indebted to Drs. Anderson and Leonard of Squibbs Biological Laboratory for carefully extracting the clotting elements of the tissues of a pair of tonsils dispatched to them several months ago. The tonsils from a child of six were removed with almost no bleeding. The clotting time of his blood was 8 minutes. The capsule was removed from the tonsils as carefully as possible and an extract made from this tissue as well as that of the gland itself. Tests were then made with sheep plasma, using varying dilutions of the tissue extracts to determine the clotting time of the glandular portion, and the capsular portion, compared with thromboplastin (Squibb). The normal clotting time of the sheep plasma, without the addition of any coagulent, was 18 minutes and 5 seconds. All dilutions were made by adding physiologic salt solution to the tissue juice extract. All tests were made in duplicate. The test was made after adding two drops of the varying dilutions of the extract to the sheep plasma. The result of the test follows:

C—CAPSULAR PORTION.

C—undiluted	1 min. 35 sec. 1 min. 38 sec.	C 1 in 500 dil.	7 min. 35 sec. 8 min. 0 sec.
C 1 in 5 dil.	1 min. 10 sec. 1 min. 25 sec.	C 1 in 1,000 dil.	13 min. 5 sec. 13 min. 25 sec.
C 1 in 10 dil.	2 min. 20 sec. 2 min. 25 sec.	C 1 in 5,000 dil.	19 min. 10 sec. 19 min. 25 sec.
C 1 in 100 dil.	4 min. 15 sec. 5 min. 35 sec.	C 1 in 10,000 dil.	16 min. 20 sec. 27 min. 2 sec.

G—GLANDULAR PORTION.

G—undilated............ 0 min. 53 sec.	G 1 in 500 dil......... 3 min. 5 sec.
0 min. 58 sec.	3 min. 40 sec.
G 1 in 5 dil. .... 1 min. 10 sec.	G 1 in 1,000 dil...... 6 min. 20 sec.
1 min. 10 sec.	6 min. 30 sec.
G 1 in 10 dil............ 1 min. 30 sec.	G 1 in 5,000 dil......11 min. 5 sec.
1 min. 35 sec.	11 min. 55 sec.
G 1 in 100 dil......... 1 min. 40 sec.	G 1 in 10,000 dil....17 min. 5 sec.
1 min. 35 sec.	18 min. 10 sec.

S—THROMBOPLASTIN LOCAL SQUIBB.

S—undiluted............ 0 min. 50 sec.	S 1 in 500 dil......... 3 min. 30 sec.
0 min. 53 sec.	3 min. 20 sec.
S 1 in 5 dil............. 0 min. 50 sec.	S 1 in 1,000 dil...... 6 min. 10 sec.
0 min. 47 sec.	7 min. 0 sec.
S 1 in 10 dil........... 1 min. 10 sec.	S 1 in 5,000 dil......12 min. 1 sec.
1 min. 15 sec.	10 min. 40 sec.
S 1 in 100 dil......... 1 min. 30 sec.	S 1 in 10,000 dil....18 min. 10 sec.
1 min. 40 sec.	18min. 40 sec.

It may be of interest to quote from Mills,[7] who has done a large amount of work on the clotting of blood. He says, "The circulating blood contains very little tissue fibrinogen, so that it is able to remain fluid and still contain soluble calcium salts and blood fibrinogen. However, in event of tissue injury with rupture of blood vessels, the escaping blood is immediately mixed with tissue juices and clotting induced. It has been found that those tissues in which hemorrhage is most dangerous possess the richest store of tissue fibrinogen, so that bleeding there is most quickly controlled. Such tissues are the brain, lungs, kidney and the endothelium of the blood vessels. Not only does the escaping blood clot quicker when mixed with the juices from the tissues but the amount of fibrin formed is much greater and the texture more firm. The additional strength thus imparted to the fibrin plug filling in the opening renders subsequent rupture less likely."

In my original paper, published in 1916[8] (Laryngoscope), I made use of the tonsil as a means of preventing hemorrhage and lessening trauma only during the first stage of the dissection of the gland. However, during the last several years I have extended the use of this technic till now I rely largely on the application of the tonsil to the fossa for the control of bleeding during the entire process of dissection and after removal. In my experience it accomplishes this purpose far better than a gauze sponge, in that it supplies the clotting ele-

ments of the tonsils and the wounded tissues to the mouths of
the open vessels, thus forming a firm and abundant clot. In
the technic of holding the tonsil firmly in the fossa before
bleeding takes place, the tissue juices are applied in the most
effective way, for the reason that these coagulent elements are
not diluted nor are they washed away by the first rush of
blood. Leaving out of consideration for the moment the action
of the tissue juices in causing clotting, there is no sponge
which would give such accurate pressure with so little trauma
as the tonsil itself. Furthermore, surgical sponges by their
absorptive action on the tissue juices tend rather to lessen
the clotting tendency of the blood, though they are effective
as a means of applying pressure. In this connection it may be
of interest to quote from a personal communication of Prof.
Howell of the Johns Hopkins University. He says, "Your
procedure can certainly be depended upon on a priori grounds
as introducing a favorable condition for clotting. The point
is the clotting of blood is much favored by tissue juice, the
socalled thromboplastic action of the tissues. In dissecting off
the tonsils there is much wounding of the tissues, but by your
procedure of laying back the tonsil you add something to this
exposure of the oozing blood to the tissue juice of the lacerated
tissue and thus help the process of clotting." The technic in
brief is as follows: The tonsil is seized with forceps and the
upper third is carefully dissected, using by preference sharp
or semisharp instruments. Should vessels of any importance
be encountered during this stage of the operation, the tonsil
is at once pushed back into the fossa and held firmly till bleed-
ing is controlled. When the upper part of the tonsil has been
sufficiently dissected the wire loop is slipped over the tonsil
and its removal accomplished by slow action of the wire snare.
The time required for this step is about one minute for each
tonsil. The last and most important step in the operation after
its complete severance is the holding of the tonsil firmly and
accurately in its fossa for a period of about two minutes. This
may be done by the assistant, and it should be borne in mind
that the gland is at no time taken from its position in the
fossa till about two minutes have elapsed to insure the closure
of the mouths of the vessels with a firm clot. As a rule,
though, of course, there are exceptions, it is not necessary on

removal of the tonsil to sponge the fossa to control any imme-
diate postoperative bleeding, and rarely is it necessary to catch
a bleeding vessel. The avoidance of sponging and the use of
forceps in the wound will greatly lessen trauma. Although
the incidence of shock in varying degrees of intensity is still
not perfectly understood, yet it is a well recognized fact that
hemorrhage and trauma are important factors in its produc-
tion. It is a well known fact that young children, and those
of all ages whose health is below par, withstand the loss of
blood badly. The fact that so many of our patients requiring
operation come within this class makes it desirable to empha-
size the importance of conserving all blood possible.

The objection which has been offered to this method of the
control of bleeding is the supposed danger of introducing the
infection present in the tonsils into an open wound. This dan-
ger appears to me very remote, for the reason that the severed
side of the tonsil is not the infected area of the gland. There
is quite as much danger of carrying infection with a surgical
sponge, for the reason that the mouth is the natural habitat of
various infectious organisms.

In conclusion, I wish to emphasize the following points:

1. The application of the tonsil itself to the open vessels
of the fossa controls bleeding more effectually than the usual
method of sponging.

2. This technic conserves the patient's blood and at the
same time lessens trauma.

3. In the reduction of hemorrhage and trauma the danger
of infection becomes less and convalescence is hastened.

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XIII.

THE INFLUENCE OF VISION ON NYSTAGMUS.

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Isaac Jones[1] has said "The intimate relation between the ear and the eye can be best appreciated when we realize that the ocular mechanism depends upon stimuli from the ear for precision of movement." The intimacy of this relation may be greater than anticipated, if we note the control that vision exerts over the manifestations of vestibular function.

There has been a great deal of discussion since the time of Flourens (1828) in regard to the cause of the compensatory eye movements, or "with-nystagmus" during rotation. When a normal pigeon is rotated (head free) certain movements of the head are noted—that is, the head seems to move slowly to the left if rotated to the right (or vice versa), and then it moves quickly in the direction of rotation. These movements are called "the with-nystagmus." Gruenberg[2] stated: A frog (on a turntable) "will always turn his head to the left if the table is turned to the right and vice versa." This does not, to my mind, explain the phenomenon. The pigeon, or the frog, does not turn his head to the left if rotated to the right. Sensations impressed upon the retina attract the visual attention, therefore the vision remains fixed for a short period of time; meanwhile the turntable to which the pigeon is attached is moving away from the line of visual fixation.

During the rotation the pigeon's body moves with the turntable while the head lags behind until there is a conscious change of vision, or until the extreme twist to the neck muscles causes the quick phase "of the with-nystagmus"—that is, a movement in the direction of rotation. Gruenberg[3] thought the with-nystagmus could be explained on the theory of a constantly changing view during rotation. Together with Wills, he thought the compensatory movements might be, in part,

due to the "spin" to which all objects are subject when rotated. His opinion was by no means certain, because he adds: "They (compensatory movements) may be quite independent of visual impressions. One is therefore driven back to the reexamination of the semicircular canal theory or to search for some other precursory movement or acceleration." Jones[4] states: "During the turning the canal is moving away from the endolymph. The current, therefore, in the right canal is toward the ampulla. The result, therefore, during the turning is a vestibular pull of the eyes to the left—that is to say, a nystagmus toward the right. It was this nystagmus during turning that was studied by the original investigators." Rejto's[5] observations are quite similar to those of Jones. We quote: "Let us, for instance, consider the cause of events when the head is revolved to the right, in the horizontal plane. The slow movements of the eyes are directed during the revolving to the left, and after the sudden cessation of the revolving to the right. The cause of these reflex eye movements are the different endolymph currents in the canals. During the revolving to the right the endolymph current is to the left in both horizontal canals, which means, with regard to their anatomic positions, ampullofugal direction in the left and ampullopetal in the right canal, after the sudden stopping ampullopetal in the left and ampullofugal in the right canal."

In performing a number of experiments on the vestibular apparatus of the pigeon it was evident that the frequency of the "with-nystagmus" of the head, or eyes, during rotation depended upon the rate of rotation. The slower the rotation the greater the number of head movements, and vice versa. These movements range from 1 to 12— in a rotation of 360°. It was noted that the eyes, per se, did not enter into the movement if the head was free to move, but if the head was stationary the eyes moved. It seemed probable that vision was partly responsible for the "with-nystagmus" on account of its variance according to rate of rotation. Vestibular stimuli could hardly produce such a variation in the "with-nystagmus." Consequently numerous methods were employed to overcome the influence of vision, without destroying the sight, but all proved unsatisfactory. Finally experimental amblyopia was attempted. The use of injections of India ink into the anterior

chamber and the cornea did not prove satisfactory. The desired results were obtained by injecting a few drops of a saturated solution of ammonium sulphate directly into the retina, which produced a permanent amblyopia without destruction of the iris, thereby making it possible to better interpret the eye nystagmus. It requires approximately ten rotations of the human to demonstrate the "after-nystagmus." A glance at protocol No. 1 shows that ten rotations, of the normal pigeon, in ten seconds, produces an after-nystagmus lasting from 2.0 seconds to 4.3 seconds. When a pigeon with experimental amblyopia is rotated head free (one revolution in ten to twenty seconds), there was no compensatory movement, or "with-nystagmus" of either eyes or head, which seems to refute the idea that the "with-nystagmus" was due to vestibular stimuli. The contention may be raised that the rate of rotation was too slow to produce any movement of the endolymph, but a perusal of protocol No. 2 shows that this is erroneous, since the same pigeon, with amblyopia, when rotated one revolution in 10 seconds, did not develop a "with-nystagmus," but on cessation of rotation developed an "after-nystagmus" lasting 10.6 seconds.

When rotated one-half turn in 10 seconds no "with nystagmus" was noted, but an "after-nystagmus" of the head lasted for 6.4 seconds. When rotated one-fourth of one revolution in 5 seconds, no "with nystagmus" developed, but an "after-nystagmus" lasted for 4.4 seconds. Aften ten rotations in ten seconds there was a "with-nystagmus" of the head and an "after-nystagmus" lasting for 12 seconds. This "with-nystagmus" was due to the head being pivoted and not able to keep up with the moving body, hence a mechanical lag of the head away from the direction of rotation. The neck "twist" disrupts the muscle balance; this, in all probability, is the stimulus that causes the quick phase of the "with-nystagmus."

Pigeon C2 (normal vision), in which the ampullæ of both horizontal canals had been removed, when rotated in the plane of these canals (either to the right or left) developed a "with-nystagmus" but no "after-nystagmus."

If we accept the theory that rotation of the normal pigeon in the horizontal plane causes a movement of the endolymph in horizontal canals which is accompanied by a "with-nystag-

mus" and following rotation a horizontal "after-nystagmus," then it must be evident in Pigeon C2 no endolymph movement could take place, since the ampullæ of these canals were destroyed, consequently we must look elsewhere for the stimuli producing the "with-nystagmus" in this pigeon.

Since pigeons with experimental amblyopia, when slowly rotated in the horizontal plane manifest no "with-nystagmus" but on cessation of rotation develop "an after-nystagmus," it trolling influence over vestibular function.

CONCLUSIONS.

It requires at least five revolutions in five seconds to produce any appreciable "after-nystagmus" in normal pigeons. Normal pigeons always have a "with-nystagmus" and an "after-nystagmus" following rotation.

With experimental amblyopia it requires only one-quarter of one revolution, in ten seconds, to produce an "after-nystagmus" of 2 seconds' duration, which (in the pigeons tested) was not preceded by a "with-nystagmus" during rotation.

Blind pigeons when rotated ten times in ten seconds developed a "with-nystagmus," which it would seem (from the experiments cited) was not due to the endolymph movement but due to the mechanical "spin" to which all bodies are subject when rotated.

Pigeons with the ampullæ of both horizontal canals removed, when rotated develop a "with-nystagmus," but do not develop an "after-nystagmus."

From the foregoing experiments one is justified in the belief that vision exerts a controlling influence over vestibular function.

PROTOCOL No. 1. NORMAL PIGEONS.

Pigeon No.	No. of Rotations	Dur. of Rotation in Sec.	Duration of Rotation	"With-Nystagmus" During Rotation	"After-Nystagmus"		
					Type	Direction	Dur. in Sec.
B1	10	10	R	Yes	Hor.	L	3.4
B1	10	10	L	Yes	Hor.	R	4.0
B2	10	10	R	Yes	Hor.	L	3.1
B2	10	10	L	Yes	Hor.	R	4.1
B3	10	10	R	Yes	Hor.	L	3.4
B3	10	10	L	Yes	Hor.	R	4.0
B4	10	10	R	Yes	Hor.	L	3.0
B4	10	10	L	Yes	Hor.	R	4.3
B5	10	10	R	Yes	Hor.	L	2.1
B5	10	10	L	Yes	Hor.	R	2.3
B6	10	10	R	Yes	Hor.	L	3.4
B6	10	10	L	Yes	Hor.	R	3.8

PROTOCOL No. 1A. NORMAL PIGEONS.

Pigeon No.	No. of Rotations	Dur. of Rotation in Sec.	Duration of Rotation	"With-Nystagmus" During Rotation	"After-Nystagmus"		
					Type	Direction	Dur. in Sec.
B1	20	10	R	Yes	Hor.	L	6.0
B1	20	10	L	Yes	Hor.	R	6.2
B2	20	10	R	Yes	Hor.	L	6.4
B2	20	10	L	Yes	Hor.	R	7.3
B3	20	10	R	Yes	Hor.	L	6.4
B3	20	10	L	Yes	Hor.	R	7.1
B4	20	10	R	Yes	Hor.	L	6.0
B4	20	10	L	Yes	Hor.	R	7.1
B5	20	10	R	Yes	Hor.	L	3.0
B5	20	10	L	Yes	Hor.	R	3.2
B6	20	10	R	Yes	Hor.	L	6.8
B6	20	10	L	Yes	Hor.	R	7.2

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PROTOCOL No. 2. TOTAL AMBLYOPIA.

Pigeon No.	No. of Rotations	Dur. of Rotation in Sec.	Duration of Rotation	"With-Nystagmus" During Rotation	"After-Nystagmus"		
					Type	Direction	Dur. in Sec.
BL1	10	1	R	No	Hor.	L	10.6
BL1	10	1	L	No	Hor.	R	10.4
BL2	10	1	R	No	Hor.	L	10.4
BL2	10	1	L	No	Hor.	R	10.2
BL3	10	1	R	No	Hor.	L	11.4
BL3	10	1	L	No	Hor.	R	12.2
BL4	10	1	R	No	Hor.	L	11.1
BL4	10	1	L	No	Hor.	R	11.8
BL5	10	1	R	No	Hor.	L	9.6
BL5	10	1	L	No	Hor.	R	9.2
BL6	10	1	R	No	Hor.	L	11.1
BL6	10	1	L	No	Hor.	R	11.6

PROTOCOL No. 2A. TOTAL AMBLYOPIA.

Pigeon No.	No. of Rotations	Dur. of Rotation in Sec.	Duration of Rotation	"With-Nystagmus" During Rotation	"After-Nystagmus"		
					Type	Direction	Dur. in Sec.
BL1	¼	5	R	No	Hor.	L	4.4
BL1	¼	5	L	No	Hor.	R	4.6
BL2	¼	5	R	No	Hor.	L	4.4
BL2	¼	5	L	No	Hor.	R	4.1
BL3	¼	5	R	No	Hor.	L	4.6
BL3	¼	5	L	No	Hor.	R	4.8
BL4	¼	5	R	No	Hor.	L	4.8
BL4	¼	5	L	No	Hor.	R	4.6
BL5	¼	5	R	No	Hor.	L	2.0
BL5	¼	5	L	No	Hor.	R	2.1
BL6	¼	5	R	No	Hor.	L	4.1
BL6	¼	5	L	No	Hor.	R	4.6

Abbreviations: R., right; L., left; Hor., horizontal.

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XIV.

THE NATURE OF THE INFLUENCE OF FOCAL INFECTION AND THE MEANS NECESSARY TO MEET IT.

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I am very sensible of the compliment you pay me in giving me an opportunity to talk to you today on the subject of focal infection. I believe, as an internist, that we internists as a group have given too little philosophic consideration to the varied syndrome which arises from this cause and to the nature of the pathology behind it. I feel that the work that you gentlemen have been doing, and also that the orthopedists have been doing, in relation to arthritis, has been of great value in bringing before us some of the more important phases of this topic. Unless we are willing at the present state of pathologic knowledge to regard focal infection from the standpoint, for example, of round cell infiltration and gross morbid anatomy alone, we are driven to the necessity of studying some of the many fields which this condition brings about. As long ago as the '80's, Charcot called attention to a large group of cases which come under this general heading, such as neurasthenia, optic disturbances, diseases of the skin, arthritis and so on. Much of what I have to say today will be grouped around one of these topics, particularly arthritis, but I do not want you to believe, if you please, that I am discussing arthritis alone, because I think nothing is more sure than that focal infection produces many conditions and that arthritis is only one of them. Indeed many of these conditions may occur coincidently in the same individual. Because of its external manifestations, however, arthritis lends itself better than the others to analysis and treatment.

In taking this question up it will be necessary, from the standpoint of the internist and biochemist, to refer to technical

matters, but I will try to be as brief as possible. I have
been exceedingly interested in the papers bearing on focal
infection that have gone before, and I shall have a great
deal to say both pro and con, but I want to make clear
at the outset that I believe focal infection to be a cause
of disease and indeed the cause of a great many kinds
of diseases. As a matter of fact, all of our cases of neuras-
thenia, arthritis, etc., were studied routinely by specialists in
genitourinary and nose and throat lines, dentistry or any other
which seemed pertinent. However, it is important to realize
in the first place that removal of focal infection, as some of
the speakers have remarked, does not always cure the dis-
ease in question. In the second place, it is important to real-
ize that some cases of neurasthenia, arthritis, etc., are appar-
ently favorably influenced by other factors, and lastly, it is
important to try to find out just what it is that focal infection
does after all. We have learned heretofore little or nothing
on this point. During the war I was fortunate in being in
charge of the intensive study and treatment of arthritis in this
country for the army, and we had the opportunity of studying
a great many cases, a larger number, indeed, than had ever
been studied before under anything approaching controlled
conditions. In this connection I want to call your attention
to these charts, which portray certain outstanding facts that
our studies developed.

We had 400 cases of chronic arthritis; and it was a great
surprise to us to find when we analyzed them for the
apparently exciting factor, that exposure stood at the
top of the list by markedly predominating figures. Nearly
60 per cent of the cases were referable to immediate-
ly preceding exposure of some kind, either in water or
from sleeping on damp ground, on stone floors or in
other ways. Dysentery came next, but much lower, and it
was interesting to note that tonsillitis was pretty well down
the list—3 per cent only. These cases were all analyzed from
every standpoint that the internist could bring to bear. They
all had complete physical examinations, blood counts, Wasser-
manns, etc. Twenty-seven per cent of cases had apparently
contracted arthritis in the absence of demonstrable surgical
foci. It is of course difficult to say that a case is absolutely

free of foci of infection. However, on the other hand, 293
cases, or 73 per cent, showed definite demonstrable surgical
foci either in the teeth, tonsils, genitourinary tract, sinuses or
a combination of these, and I think there is very small room
for error on this side. If anything, the number was higher rather
than lower. Two hundred eight cases or 52 per cent of the entire
series showed tonsillar infection; 134, or 33 per cent, showed
dental infection; 12 per cent showed genitourinary conditions:
20 per cent showed a combination of these. Now, the point
which I think will perhaps interest you most, as it did us, and
was indeed very surprising to us, is brought out in the follow-
ing figures: Twenty-three per cent, 92 cases, recovered in the
apparent absence of any surgical focus. One hundred eighty-
four cases or nearly one-half of the entire series, however,
recovered in the presence of a demonstrable focus of infec-
tion. This means that a great many men came into the hos-
pital well or they came in convalescent and got well, or they
came in and recovered after treatment other than opera-
tive. But at all events, nearly one-half, or 46 per cent,
got well in the presence of a definite surgical focus. Ton-
sillectomy was one of the most frequent procedures carried out
in the way of removing focal infection, and about 16 per cent
improved or got well as the result of removing surgical foci
in the seven months that we had opportunity to observe these
men. So that in the last analysis, looking at the matter dis-
passionately and desiring simply to get facts, the outstanding
deduction is that an overwhelming proportion got well in the
presence of demonstrable surgical infection, and relatively few
got well as the result of removing foci. It is perfectly true
that these cases were nearly all in young men between the ages
of 20 to 32 and that they were more or less selected subjects.
Their disabilities had been developed under war conditions and
constituted almost a laboratory experiment, so it is not fair
to regard these men as comparable in all respects to cases in
civil life. Nevertheless, the lesson forced upon us is that
great emphasis must be given to the fact that these men got
well in the presence of definite foci of various kinds.

We endeavored to study this topic of arthritis, or rather
the whole syndrome, from a number of different angles, and
among other things we took up the subject of basal metabol-

ism. By basal metabolism we mean the amount of heat generated by a unit of body surface per unit of time or, in other words, the intensity of the sum total of the combustive processes of the body. We found a slight reduction below normal in 20 per cent of these cases.

We also studied the urea of the blood and the normal values found give no color to the conception that the nitrogenous metabolism is disturbed and that the widely practiced avoidance of "red meat" has any justification in fact. We found some increase in the blood creatin of half the cases studied. The importance of this is that it has some relation to the carbohydrate metabolism. Apropos of the carbohydrate metabolism, we came across some other facts of rather more interest. I do not know whether you gentlemen are familiar with the socalled glucose tolerance test. Like most new measures introduced into medicine, it has been carried often to extremes, but it reveals part of the pathology back of a number of conditions. There is nothing specific about it, however, even for diabetes, where it has its widest application. One gives 100 grams of glucose by mouth and examines the sugar in the blood; before the glucose is ingested the sugar in the blood will be found at a certain fixed normal level. If, in half an hour after ingestion of the glucose, the blood is examined again, the glucose will be found to have risen to a certain amount, and an hour later it will have probably returned to about its former level.

If one studies the group of arthritics, however, a different picture is found. Using the same base line here for both groups, the curves for the arthritics go very much higher. In other words, the glucose in the blood is not removed as quickly as it should be. It may reach very high figures, almost diabetic. It is obvious that something is going on, that we do not completely understand, which interferes with the normal removal or utilization of the ingested sugar. This abnormality is not a specific disease condition per se, but some interesting things can be developed from it. I want to call attention to the case of a boy, 19 years old, with severe arthritis of the hip, who had been sick nine months. He had a very bad pair of tonsils. We determined his sugar tolerance when he came into the hospital, and we found the curve resulting was

very high. He tolerated sugar poorly and hence had a high
curve. We removed his tonsils two weeks after admission;
two weeks later he was entirely free from all arthritis and his
sugar tolerance was at a normal level.

Another case illustrates the same sequence of events in a
soldier presenting acute arthritis of wrists and knees and
ankles. The chart portrays a marked elevation of the glucose
tolerance curve and also the fact that it is not modified by doses
of salicylates, even though they controlled the symptoms. The
difference in the curves is practically negligible. This man
had his tonsils removed and two weeks later his tolerance
was down almost to normal. Here is the case of a man who
had no focal infection, at least any such had been removed
long before we saw him, though he was exceedingly sick and
confined to bed. He made a recovery from arthritis along dif-
ferent lines, dietetic lines, and it is of interest to see that while
he had a lowered sugar tolerance during the height of illness,
when definitely convalescent it again came down precisely as
after removal of the tonsils in the other cases.

A man suffered from a severe spondylitis, in whom the sugar
tolerance returned towards normal after the removal of six
abscessed teeth. Here is a case in which recovery and a return
to normal of a lowered sugar tolerance were brought about by
repeated injections of nonspecific protein, the man having no
focal infection. I present this chart, finally, which is the chart
of three individuals who had enlarged tonsils without symp-
toms and were apparently active, healthy men. Two of them
gave normal curves, a third slightly elevated. They had no sys-
temic symptoms. The man who had a higher curve had pre-
viously given a slightly high curve in other hands. Something
evidently was going wrong in his systemic metabolism.

Consideration of these general findings led us to wonder
what was going on to produce these results. The work of the
English physiologists, and subsequently the American, has
drawn attention to the blood gases and the respiratory func-
tions of the blood. For a number of reasons, which I won't
take your time to consider, we took up the study of the blood
gases in this syndrome, of which arthritis is one expression.
We found that if the blood gases of a vein of the arm were
studied by a given technic which we used, one got figures

for the oxygen percentage saturation of the blood corresponding to about 42, but if we studied the arthritics we found somewhat higher figures, which were roughly in the neighborhood of 54, some much higher. This means that apparently as the blood comes back from the periphery there is, under the conditions of these observations, a tendency for the blood to contain more oxygen than is the case in a normal individual. For some reason or other it is apparently not removed as it normally is, possibly because the tissues cannot utilize it, but more probably for another reason, to which I will refer later. It is also interesting to note (and this is rather corroboratory evidence) that if one studies the blood during the conduction of a test revealing a lowered sugar tolerance, an increase in the percentage of saturation of oxygen is found, parallel with the rise in the concentration of sugar, and yet these changes do not seem to be due to the same mechanism. Sometimes the blood gases change more evidently than does the sugar tolerance during this test. We tried to explain it on a chemical basis. We could not find, however, that the blood held on to its oxygen more than it should in a strictly chemical sense. We were therefore driven to a consideration of the possibility that the blood went by the tissues so fast or in such a way that the oxygen could not be removed. It is well known that in conditions of hyperemia this is the case. If one studies persons during hydrotherapy, or an electric bake, the oxygen of the peripheral blood shows the same changes, and it is practically certain that the blood is going by faster under these conditions. This led us into a further study of bakes, hydrotherapy and the application of heat at large, and we have found some rather interesting results. I want to take time to call your attention to them because these measures are used widely in medicine. They are as old as Hippocrates and are employed by many different groups of physicians, such as neurologists, surgeons, ophthalmologists and internists. They have wide application in the treatment of nephritis, arthritis and the insanities. We have found if one studies the sweat under these circumstances that some interesting changes take place. The sweat normally is about neutral, though it may be actually acid or slightly alkalin, but whatever it be, during the process of hydrotherapy or the application of external heat of any kind,

the sweat changes its reaction. If it started acid, it becomes
neutral; exceptionally it may swing all the way from mark-
edly acid to markedly alkalin, but it never stays the same,
always changing towards the alkalin range. Ninety-nine per
cent of cases and all normals undergo this very definite change.
A few cases of marked arthritis have not changed in their
figures at all, remaining acid throughout. Now, carrying this
further, we have also found that a good many other things
take place during the course of one of these treatments,
though there is not time to go into the chemistry in detail,
much as I would like. Dr. Cajori and Miss Crouter, working
in my laboratory, have found and recently reported the fact
that there is a rise in the carbon dioxid dissociation curve of the
blood. Direct measurements also show a change in what we call
the hydrogen ion concentration, or the reaction of the blood,
which becomes more alkalin during this procedure than it was
before. This is entirely in keeping with the change which the
sweat shows, and it is also accompanied by changes of a parallel
nature in many cases in the urine. So there is apparently
something in the nature of an alkalin wave during these pro-
cedures. It is interesting to note during the study of these
individuals that if one puts a rubber bag around the arm, and
the individual is subjected to a "bake," the air in the bag will
be found to increase in its carbon dioxid content. There is
undoubtely a loss of carbon dioxid through the sweat and
through the lungs. This loss of acid (CO_2) causes the blood
to become more alkalin, and the excess of alkali is then in turn
excreted through the sweat and urine. It is important to
remember that carbon dioxid is an end product of metabolism.

Because of the fact that we cannot explain all of the above
changes in the blood entirely on chemical grounds, we were
driven to study the question of blood flow, making use of the
hand calorimeter. This is a jar or box, containing water, into
which the hand was put with the aim of ascertaining the in-
crease in temperature of the water produced by the blood. We
were able to show that the arthritic tends to have a little less
rapid blood flow than the normal individual. That led us
again to consider the question of the capillaries, to which a
great deal of attention has recently been drawn, and it is pos-
sible to demonstrate in a few cases of arthritis the fact that

in certain regions, such as the finger, they are finer and less
conspicuous than in normals. The evidence on this point is
by no means final, but it is at least suggestive and fits in with
what I have already mentioned to you.

In other words, there seems to be evidence to indicate that
in this syndrome there is a disturbance of metabolism, and
that it is a little below normal, that there is an impairment
of oxidation in a local and possibly a systemic sense and that
this disturbance arises from the way in which the blood passes
through the tissues. This is a very cursory survey of the facts
relating to the dynamic pathology of this condition as we
see it.

It is of interest to note in studying arthritis that the agents
that are of value in meeting the symptoms are those that im-
prove the local or systemic metabolism or the local or systemic
blood flow, and it has been of interest to correlate that with
the findings which we have obtained in our work. Much has
been said today about the removal of focal infection, and I am
entirely in accord with the view which advocates the removal
of focal infections of various kinds when this can be done
without too great and extensive damage to the individual and
in suitable cases. Sometimes, however, the results do not fol-
low which we anticipate, and I think it is of the highest im-
portance to bring out the fact that treatment of the arthritic
or other member of the group under discussion is not neces-
sarily complete with removal of a surgical focus of infection.
A great many cases hang fire after that point. If they are
regarded as referable only to this case, and if the subsequent
pathologic disturbances which may arise are disregarded, the
individual may remain in poor health for a long period of
time or permanently, whereas if one takes a broader stand-
point it is frequently possible to bring the individual back to
health. Following long standing focal infection there is
a more or less permanent dislocation of some physio-
logic functions as can be shown by these charts. In some
cases with a marked dislocation of the sugar curve this
could be shown to come down progressively as they con-
valesced, and it is obvious that if two, three, four or six
weeks are required to bring it down, by the same token as
many months may be required for the return, and sometimes

it does not return at all.   Now, some of the agents which in
general are of value in this condition towards improving
metabolism are the following : Arsenic, radium, potassium iodid,
thyroid extract, and it is of interest to note that Kendall, the
discoverer of thyroxin, states that it acts by virtue of CO_2.
which it picks up and carries out of the body.

Of course the socalled radium waters and emanators
commercially exploited are in general of small use because
there is very little radium in them.   The X-ray has the
same influence.   Massage is also of value, as is exercise in
general.   The value of nonspecific protein in this con-
dition is probably almost solely due to the fact that a tre-
mendous increase of metabolism arises from its use, accom-
panied by fever and some other phenomena.   The use of
vaccines I shall not refer to, because you are all familiar with
them.   They have no specific value or action upon the re-
sultant arthritis or neurasthenia, per se, so far as I am aware.
I think it is important in considering this general question and
the operation of focal infection to appreciate also the other
factors which bring about the same syndrome and produce
analogous results.   The gastrointestinal tract is a field which
is productive of a great deal of damage, and attention has
been very properly drawn, by the Boston School of Ortho-
pedists in particular, to ptosis, malfunction and other disturb-
ances which undoubtedly contribute to absorbable toxins.

I would like then to present to you the thought that arthritis
and this general syndrome may be regarded as part of a sort of
metabolic equation.   On the one hand, we have the elements
or factors which make for a diminution in metabolism and a
variety of clinical symptoms ; on the other hand, we have a
variety of agents which improve metabolism, and if these fac-
tors can be properly equilibrated or balanced against each
other. one may have absence of symptoms, and that is precisely
what is often taking place in the individual during health.
A lot of people have focal infections, possibly many
people here today have them, and are not sick.   It is
important to note that this general relation has been
known for generations and is the real raison d'etre for
the Spas and "Kurorts" to which patients flock by the
hundreds of thousands.   Many individuals go to these

places and get well. I think, as a matter of fact, that we are most of us to blame for not realizing that there are many ways of relieving this disturbed physiology. By removing or replacing the first link, a focal infection, say, we do not necessarily restore the chain in its entirety. That is the reason why these institutions succeed. I am sure you have all had illustrations that it is possible to effect great improvement in the clinical condition of these sufferers by reason simply of the improvement in their general physiologic processes, and I mean by that something more specific than outdoor air, good food and early retiring. I mean such measures as massage, sweating, exercise and drugs, and so on. Before leaving this question of the general "balance of health" and the factors that make for ill health or improvement I want to call attention to some interesting observations that were made by the English physiologist Barcroft, who found that at great altitudes some of the symptoms arise which the arthritic and neurasthenic suffer from peculiarly; for instance, headache and fatigue. There are even certain changes in the ends of fingers which resemble hypertrophic pulmonary osteoarthropathy. In some of these conditions the actual bone is concerned. The relation of oxygen want to these conditions is clearly proven.

I have spoken of the gastrointestinal tract and the fact that if the anatomy or physiology is wrong various consequences arise. They arise, in part at least, from the fact that the food is not properly taken care of and toxins are formed, probably from retention. There is another way, however, in which the food intake may operate in a more specific sense. If it is true that there is a difficulty in arthritis in completing some phases of the oxidative metabolism and if it is true that agents which improve this metabolism are helpful, then it should follow that measures designed to lighten the load on that metabolism should be of value. This is indeed apparently the case. If one reduces the food intake in these individuals to a point considerably below that they have been accustomed to and gives the body less to metabolize, brilliant consequences sometimes follow. For example, one of these sugar tolerance charts is that of an officer who got well along dietetic lines. He was eating 3,500 calories a day and lying in bed all day. That is a large amount of food, which only soldiers in service

or workers in the Maine woods can utilize. We put him on 2,100 calories on the basis of the thought that we could spare his metabolism an unnecessary burden. A very interesting result followed. In a few days he was able to get out of bed, in a few days more he dispensed with crutches and he eventually left the hospital, symptomatically well.

I merely mention this because it is illustrative, and rather graphically, of the improvement which may take place when one considers the metabolism of these cases. I may say that this work has recently been corroborated by Fletcher, of Toronto. In a large series of 150 cases, he found that about 40 per cent of unselected cases showed improvement from these measures without regard to whether or not they presented focal infections.

I do not want to be interpreted as advocating the rash use of dietetics. The real lesson to be deduced is that these cases should be regarded as open to treatment from a number of different angles, and if we combine these forms of treatment we will sometimes get results we could not otherwise achieve.

Now, this dietetic form of therapy sometimes makes a very useful basis from which to start. It is no panacea, and so far as I know there is no panacea in arthritis or in the syndrome we are discussing; perhaps there is none in medicine. Some cases do not get well along this line. The cases to be so treated should be very carefully and critically chosen. Such therapy can often successfully be made the basis of other forms of treatment. One man, for example, in the service made a 50 per cent improvement and then hung fire. He had had his tonsils out some weeks before, without improvement. We then gave him hydrotherapy in addition and he made a complete convalescence and was symptomatically well when he left the hospital.

There is another phase of the dietetic treatment of these conditions which was brought out by Mendel of Yale some years ago, and by Howe in Boston, working in the Forsythe Dental Infirmary, that in a deficiency disease, such as scurvy, where the diet is unbalanced, infection may arise as a result and not as the cause of the systemic disorder. This is well illustrated in the dental caries which these cases show.

I want to bring out one other fact, and that is that some-times processes which are started by the tonsils or teeth may be perpetuated by the intestinal tract and can be reached then only by diet in the way I have tried to describe.

In closing, gentlemen, I would like very much to leave with you the conception that we should try to regard the syndrome precipitated by focal infection from a broad standpoint. I believe that focal infection does a great many things, that it produces a variety of diseases, but I also feel that the ex-treme view is nearly always a dangerous view and that it is easy to lose one's balance in this field. I don't believe myself that there is any single panacea for the general syndrome which results so often from focal infections, among other causes, and until we lose such undue enthusiasms as we are all subject to along these lines we will continue to meet with many failures. I am sure that with inadequate effort to in-clude the various measures at our disposal, failures are much more likely to result.

2120 SANSOM STREET.

XV.

THE USE OF VUCIN (ISOCTYL HYDROCUPREIN DI-HYDROCHLORIDE) IN INTRACRANIAL AFFECTIONS.

By Alfred Lewy, M. D.,

Chicago.

Now that vucin is commercially available in the United States, it having been recommended as a curative agent in septic meningitis by various clinicians in Germany,[1][2][3][4] as well as in other septic conditions, I am reporting herewith eight cases of intracranial infections in which vucin was used by me or under my direction, together with some suggestions as to further experimental use of this product.

In 1917, Kolmer and Idzumi[5] reported the use of optochin (ethyl hydrocuprein hydrochlorid), a cinchona derivative, in experimental septic meningitis in rabbits, and in 1920 Kolmer[6] published his clinical conclusions, indicating that it had at least preventive value if used within four or six hours (intrathecally) of the invasion of the meninges. When, therefore, German clinicians reported cures of what appeared to be septic meningitis by the intrathecal injection of vucin, also a cinchona derivative, claiming that it has no greater toxicity for human beings than has optochin, but at the same time a laboratory germicidal value four times as great (it was claimed to be deadly for streptococci in 1/80,000 dilution), I determined to try it, and succeeded in obtaining a small supply from Germany.

I had previously tried bile salts intraspinally on a dog, hoping to find something efficacious against pneumococci (bile salts are used in the laboratory to dissolve pneumococci), but 3 cc. of a 10 per cent solution caused so severe a collapse, with marked fall of blood pressure, lasting 50 minutes on the kymograph record, that I did not care to try this agent further. Four cc. of vucin 1/1,000 in 0.4 per cent salt solution injected into the cisterna magna of a dog caused no serious

disturbance, but when I added 2 more cc. there was a sudden drop in blood pressure, and artificial respiration had to be resorted to. Lumbar puncture with withdrawal of 3 cc. of spinal fluid relieved this condition, so it seems likely that it was due to too much fluid rather than to a toxic cause.

When dissolved vucin is said to lose its germicidal value rapidly. The supply obtained by me was in tablet form, in which condition it is said to be stable, and fresh solutions were prepared for each injection. Linck[1] advocated 10 cc. of a 1/500 solution in normal salt, intraspinally, daily if necessary, as a curative dose, or 10 cc. of a 1/1,000 solution as a preventive dose, in intracranial operations or injuries where meningitis might be feared as a sequel. Some experimenters stated that in the 1/500 dose there was sometimes caused a transient paralysis of the bowels or bladder, and in one of my cases (No. 2) this condition appeared following the injection, but seemed to be relieved promptly by eserin 1/150 grain hypodermically. As this was a case of cerebellar abscess the bowel inaction may have been due to that alone and not to the vucin.

Both in Ringer's solution and in normal salt solution at body temperature and even warmer, vucin forms a milky solution, becoming more cloudy on cooling. It forms a clear solution in alcohol and in hot distilled water. That was my reason for using 0.4 per cent salt solution as a vehicle. Until experimentally proven safe, I did not want to use the distilled water or acidulated salt solution.

How much of the curative value is lost in the milky precipitate is at present conjectural, and what changes take place in contact with living spinal fluid, both normal and purulent, is also unknown. H. Birkholz[7] found that the vucin could not be demonstrated in spinal fluid withdrawn eight hours after injection, but that it does not disappear from spinal fluid kept in vitro. He believes that it combines with the salt, globulin and cells and is absorbed, and therefore cannot have much germicidal value in vivo. He also calls attention to the difficulty of diffusion of fluid introduced either by lumbar puncture or subarachnoid irrigation, and is rather skeptical of its clinical value. That it loses its germicidal value on standing in all but alcoholic solutions is admitted by its advocates.

Analysis of the Cases.—Linck[1] reported ten cases of clinical meningitis with six recoveries, but of only one of these cases, No. 8, does he state that a positive culture was obtained from the spinal fluid, in this case staphylococci. Zimmerman reports a case of cerebellar abscess, drained, with meningitis developing ten days later; spinal fluid at first sterile, but later, (at which taking is not stated) diplococci were found. The patient recovered. Kurt Huenges[3] reports a case of violent clinical meningitis, with diplococci in the smear of the spinal fluid, no culture report, one injection of vucin, with recovery.

Of the cases herewith reported by me, all showed definite clinical evidence of meningitis—that is, headache, vomiting, stiff neck and cloudy spinal fluid containing increased neutrophiles. Case 1, pneumococcus cultured from the spinal fluid, was in extremis when seen, with fixed pupils, paralysis of one arm and complete coma, and died in a few hours.

Case 4, with a definite clinical meningitis, had a sterile spinal fluid, although a high cell count, at the first taking. The second taking of fluid developed streptococci in spite of the previous injection of vucin, and the patient eventually died, after a slight remission.

Case 5 was a generalized streptococcus meningitis, presumably by way of the labyrinth, with death.

Case 6, a generalized streptococcus meningitis, probably hematogenous, and possibly associated with brain abscess. Result fatal.

Case 7, a generalized streptococcus meningitis following late after drainage of temporosphenoidal abscess. Fatal.

All of the cases which showed a positive culture from the spinal fluid eventually died. In cases 1, 5, 6 and 7 the meningitis was well diffused before the vucin was used. In case 4 it was used earlier but failed to prevent the fatal termination. Because of the apparent remission and the sterile first culture, I did not repeat the injection of vucin the following day, and also because the previous case, No. 3, reported below, did so well on one injection.

Case 3, a well developed clinical meningitis, 3,000 cells in the spinal fluid and some cocci in the smear at the first taking, increasing meningeal symptoms and 7,000 cells in the second taking of spinal fluid, but with sterile culture, fully re-

covered after one injection of vucin. This was our best case and leads us to hope that vucin may have some value as a preventive when the meningitis is still localized, or at least under conditions when the meninges may be threatened with infection, if I may use that term.

Case 2 had a working diagnosis of meningitis. After several days of observation abscess was suspected, but on account of the precarious condition of the patient and our inability to localize the lesion adequately, counsel advised against operation. Death was due to medullary compression in the foramen magnum.

Postmortem revealed an abscess in the left cerebellar hemisphere about 6 cm. behind the left ear. Left tympanic cavity showed no inflammation and was sterile, as was the abscess and spinal fluid. The abscess wall tissue showed neither syphilis nor tuberculosis.

Case 7.—Left temporosphenoidal abscess; operation and drainage May 19, 1923. Death from streptococcus hemolyticus meningitis July 20, two months later. The time of onset of the meningitis is uncertain, but there was quite a time after drainage of the abscess that patient appeared to be doing well. Death took place on the same day the vucin was used, when the meningitis was already well advanced.

Case 8.—Temporosphenoidal abscess; drainage; clinical evidence of meningitis, but culture sterile. At the date of this writing, three months after operation, patient is doing well.

Summary.—All the cases under my observation which showed a positive culture of living bacteria from the spinal fluid resulted fatally. This I consider the crux of the situation. Until we have cases which recover from clinical symptoms of meningitis plus positive culture of bacteria from the spinal fluid, we cannot be certain that our remedial agents are effective. True, nearly all the cases were treated with the vucin rather late, as they came into my hands late. Possibly earlier recognition and treatment might bring better results. I intend in future to treat diffuse septic cases of leptomeningitis by injection of the cisterna magna or by subarachnoid irrigation with vucin solution, somewhat after the suggestion of Eagleton,[8] if further experiments indicate that the procedure is safe.

Case 3 was in our opinion a localized or protective meningitis, and it is possible that in this case and in cases 2 and 8 diffusion of infection throughout the meninges was inhibited by vucin. As clinical meningitis with sterile spinal fluid often recovers without intraspinal medication no conclusions can be drawn, at least not until we have a considerable number of such cases for comparative study. In the meantime I believe we are justified in the use of vucin intraspinally in conditions which threaten the meninges with infection, for instance, operations for labyrinthitis, brain abscess, cranial or spinal trauma and infections elsewhere which threaten the meninges by metastasis.

I wish to thank Doctors Noah Schoolman, Hugh R. Schofield, Richard Street, Robert Sonnenschein, Norval Pierce, Joseph Beck, Harry Pollock and C. F. Yerger for their cooperation or the reference of cases, enabling me to make the investigations reported above.

Case 1.—(Courtesy of Dr. Noah Schoolman, April, 1922.) Male, age 53. A week ago had two intranasal operations within two days on account of headache. The exact nature of these I was unable to ascertain except that some growths, probably polyps, and some bone was removed to improve nasal drainage. The headache increased, fever supervened, and Dr. N. Schoolman was called in. He diagnosed meningitis and asked me to try the injection of vucin. When seen by me the patient was comatose, pupils unequal and nonreacting, the right arm flaccid, though patient tossed and moaned constantly. Spinal puncture, fluid canary yellow, under pressure; 50 cc. removed, which deposited about 2 cm. of heavy precipitate in an ordinary test tube. Culture showed pure pneumococci. Patient died a few hours after withdrawal of fluid and injection of 10 cc. vucin 1/500 solution.

Case 2.—J. B., male, age 53. Admitted to Hahnemann Hospital June 7, 1922. History: Ten weeks ago occipital headache began; had had pain left ear two weeks; ear was opened twice, but family are disagreed as to whether or not any discharge ensued. Several years ago had an injury to the neck which required treatment for several months, with wearing of splint. Three days before admission to hospital began vomit-

ing, which ceased after one day. (Report of Dr. H. R. Schofield.)

Present condition: Seems to be in pain; delirious, but puts hand to occipital region; neck rigid, pupils unequal, but react. Dr. Schofield reports they were equal two days before when he saw patient. Vessels of face show atheroma. Chest, abdomen negative. Scar of operation for traumatic hydrocele. Reflexes normal. Membrana tympani both reddened, left worse. Paracentesis left by Dr. Lewy, only blood obtained. Temperature 99 on admission; 102.4 in few hours; pulse 75 to 85.

Lumbar puncture, fluid under pressure and flocculent; 30 cc. withdrawn; vucin 10 cc. 1/750 injected; spinal fluid 1.250 cells per cm., 80 per cent polynuclears; culture negative after 36 hours. Wassermann negative.

June 8. Comatose; pupils small, unequal, react slightly. No plantar or patellar reflex right; Babinsky left; both arms and legs flaccid. Temperature over 102 all day. Working diagnosis: Meningitis, type to be determined. Fundi negative.

June 9. Stuporous, but can be aroused and recognizes family by name. Tongue protrusion slow and tremulous. Breathing labored at intervals. Temperature 101 A. M., 98.6 P. M., pulse from 90 to 65.

June 10. Looks better. Good hand grip. Pupils equal and react. Temperature 99.2, axillary, pulse 65 to 70.

June 11. No change. Temperature 99.6, pulse 70 to 80.

June 12. Increased headache. Spontaneous horizontal nystagmus of short excursion, to right on slight deviation right; to left on slight deviation left. Pupils unequal. Doubtful adiadokokinesia left. Heart and lungs normal. Question of left cerebellar abscess. 15 cc. spinal fluid withdrawn; 7½ cc. vucin 1/750 injected.

June 13. Examination by Dr. M. Solomon: Confused, restless, but recognizes family, hears and obeys requests; frequent spasms of pain indicated at occipital protuberance, but follows commands during these periods of pain. Right pupil larger than left; both react slightly to light; occasional nystagus on looking to left; difficulty in turning eyes upward. No tenderness mastoids or occiput; no paralysis anywhere; left knee jerk barely obtainable; right better; plantar reflex barely present; no Chaddock, Oppenheim or Babinski.

Gordon present on both sides. No neck rigidity or Kernig. Sensation not tested, patient tired out. Conclusions: Meningitis present, probably localized. No localizing symptoms justifying operation at this moment. 10 P. M. Condition unchanged; temperature 99.2, pulse 80-60. About 1 A. M., patient began Cheyne-Stokes respiration and died at 1:35 A. M.

June 7. White blood count, 13,000; neutrophiles, 76; small mononuclears, 18; large, 6. Urine, albumin trace; hyalin and granular cast. Sample taken next day showed trace of albumin only.

X-ray of mastoids, nasal accessory sinuses and neck negative.

Postmortem (head only permitted) showed cerebellar abscess left lobe, 6 cm. behind left ear; no meningitis. Spinal fluid and abscess pus were both sterile, as was culture from left tympanic cavity, which showed no pathology. Section of abscess wall showed no evidence of syphilis or tuberculosis

Case 3 (Courtesy of Dr. Robert Sonnenschein).—B. D., female, aged 6½ years; admitted to Sarah Morris Hospital October 22, 1922. History: Ten days ago discharge began from right ear; treated by a physician; gradually worse until day before admission, when fever and pain in mastoid developed. Past history measles only. Examination: Discharge right ear; pain and tenderness right mastoid; neck marked rigidity; no thyroid enlargement; no adenopathy; lungs, heart, abdomen, external genitals negative; Kernig and Brudzinski positive; reflexes exaggerated.

Diagnosis: Acute suppurative mastoiditis; septic meningitis (?).

Operation by Dr. Sonnenschein; spinal puncture; mastoid exenteration; uncovering dura; sinus accidentally opened. Mastoid contained large abscess cavity.

The spinal fluid was cloudy; 2,000 cells per cm.; globulin, + +, polynuclear cells predominate; no organisms. The pus from mastoid grew pure streptococci.

Oct. 23. Chilly, severe headache; very excitable; another spinal puncture; fluid cloudy and under pressure; temperature 104.4. This fluid showed globulin + +; 7,000 cells per cm., mostly polynuclears; a few cocci in smear but no growth in

48 hours on culture media; 10 cc. 1/750 vucin injected. At
9 p. m., an hour after spinal puncture, headache relieved, and
at 9:30 she was asleep. Temperature, 101-104.

Oct. 24. Slept well; playful. 25th. Some restlessness dur-
ing night; slept much during day; temperature 99-102.4.

Oct. 28. Severe frontal headache and pain in neck; chilly:
later felt better; temperature 100.6-103.6.

Oct. 29. Slept well; temperature 100.8-102.

Oct. 30. Restless, irritable; severe headache; temperature
101-104.6.

Oct. 31. Slept well; temperature 99-101.

Nov. 1. Slept well; some frontal headache; temperature
97.2-99.

Nov. 4. Severe headache, not long lasting; temperature
to 101.

Nov. 6. Several emeses; temperature 98.8-105.6.

Nov. 7. Slept after 10 p. m.; temperature to 103.8.

Nov. 8. Slept well; temperature to 101.2. Thereafter tem-
perature remained normal.

Nov. 12. Up in wheel chair; Nov. 15, walked a little; Nov.
21st, discharged from hospital. Recovery from now on un-
eventful, except that March 10, 1923, a mastoid fistula was
curetted and healed. Child was in good condition.

Nov. 6. On occurrence of emesis and high temperature,
examination by neurologist (Dr. Sidney Kuh): Fundi neg-
ative: pupils, wide, react; no nystagmus, no paralysis external
eye muscles; no neck rigidity, but slight resistance to turning
right; abdominal reflexes equal, brisk; Trousseau marked; all
deep reflexes within normal; muscle tone normal; knee jerks,
achilles slight but present; plantar brisk; no Babinski, Kernig,
Gordon or Oppenheim; no hyperesthesia.

White blood cell count: Oct. 22, 37,500, 87 per cent neutro-
philes; Oct. 23, 30,500, 86 per cent neutrophiles; Oct. 25,
17,500, 77 per cent neutrophiles; Oct. 27, 14,400; Oct. 28,
10,600, 68 per cent neutrophiles; Oct. 30, 12,900; Oct. 31,
8,600; Nov. 6, 11,600, 74 per cent neutrophiles; Nov. 8, 7,900,
69 per cent neutrophiles. Urine: Oct. 22, acid, albumin
+ + +, acetone + + +; Oct. 26, albumin trace, acetone 0;
No. 4, albumin 0, acetone 0.

Case 4.—L. H., female, age 6 years 10 months, referred by Drs. Street and Rupert. Admitted to Hahnemann Hospital No. 12, 1922. History: Influenza three years ago, followed by persistent cough, which has improved last one and one-half year. Measles two years ago. No previous ear trouble.

Cold in head three weeks ago; one week ago pain right ear, followed by discharge next morning (Monday). Fever 99-100 all week. Friday night cried with severe frontal headache and pain left ear. Since Saturday morning fever 103-104, vomiting; since this morning pain neck and occiput, with retraction of head. Paracentesis this morning by Dr. Street, who had been called in. No vomiting today; restless; no delirium.

On admission: Expression of apprehension; emaciation; heart and lungs negative except relatively feeble respiration left base; neck rigid to forward flexion; Kernig present, more marked right; Babinski absent; knee jerk right absent; left feeble.

Hearing present in both ears; Weber to right; postnasal discharge; tonsils negative; cheesy discharge right ear had to be irrigated out; membrana tympani gray; thickened. Left membrana tympani shows blood crust of recent incision only; no redness or swelling.

Provisional diagnosis: Acute suppurative otitis media right; meningitis—type to be determined.

White blood count, 17,500; neutrophiles 88 per cent, small lymphocytes 12 per cent.

Spinal puncture: Fluid cloudy and under pressure; 15 cc. withdrawn and 9 cc. vucin 1/1,000 injected.

Operation: Exenteration right mastoid, uncovering dura of middle and posterior fossæ; some mucopus from antrum. Deeper bone red and softened; no necrosis. Three xeroform drains.

Spinal fluid, 6,100 cells per cm.; polynuclears 90 per cent; no bacteria in smear or culture after 36 hours.

Nov. 13 to 16. No special change in condition except relief of headache and restlessness.

Nov. 16. P. m., temperature 104.2; increased rigidity; spinal puncture; 18 cc. fluid, 14 mm. on manometer; 9 cc. vucin

1/750 injected. This fluid developed streptococci with greenish reaction on blood agar; 6,300 cells per cm.

Nov. 17. Asked for nail file and manicured own nails without apparent difficulty. Temperature 101-104.

Nov. 18. Increasing restlessness and fever. Spinal fluid cloudy, 25 cc. withdrawn, 9 cc. vucin 1/600 injected.

Nov. 19. Worse. Nov. 20, stuporous; coarse rales at times. Nov. 21. A. m., died.

Blood cell counts: Nov. 12, 17,600, neutrophiles 88 per cent; Nov. 15, 14,500, neutrophiles 91 per cent; Nov. 17, 15,700, neutrophiles 89 per cent.

Case 5 (Courtesy of Dr. Norval Pierce).—H. Mc. M., Nov. 21, 1922. History: Earache right eight weeks ago; paracentesis right by attending physician same day. Second paracentesis four weeks later; in the interim discharge from ear profuse and continuous. Pain over mastoid and frontal region. Mastoid antrotomy by another specialist four weeks ago. Patient's general condition was good, and she was up in chair after operation, but frontal headache continued. Five days ago was awakened in the early morning by an attack of dizziness, nausea and vomiting. This continued every half hour for some hours with increased headache. Patient seen by Dr. Pierce three days ago. At this time temperature normal, pulse 90. She was lying on left side and had a pronounced rotatory nystagmus to left. The a_1 fork not heard in right ear. Headache, nausea and vertigo were present. No stiff neck, Kernig or Babinski. Reflexes normal; spinal fluid withdrawn was clear but under some pressure; no microorganisms in smear and culture was sterile. Culture of ear discharge showed staphylococci. Symptoms became worse and she entered St. Luke's Hospital Nov. 20 at 3:30 p. m. There is now nausea, vomiting, vertigo; nystagmus to left present but lessened; marked neck rigidity; Kernig present, Babinski absent. Diagnosis: Right suppurative mastoiditis, septic leptomeningitis (streptococcus mucosus capsulatus).

Nov. 21. Worse; stiff neck increased; pain greater; sensorium less clear.

Operation, Dr. Norval Pierce: Enlarged former incision; mastoid filled with succulent granulation tissue; curetted; bone removed in all directions as far as pneumatic spaces ex-

tended; radical operation completed; softening above and behind horizontal canal; on removal posterior canal can be seen (probable point of invasion of labyrinth), inner table corresponding to tegmen antri removed; bony wall over sinus removed; sinus wall normal color, thickness and easily compressible; promontory removed, horizontal and posterior canals opened; bone of Trautman's triangle also removed. Spinal puncture (Dr. Lewy), cloudy fluid, under pressure; 100 cc. withdrawn and 10 cc. vucin 1/500 injected into the canal.

Nov. 22. Partial facial paralysis right; quiet and restless by turns; moans less and cries out not at all; some delirium. Neck unchanged; Kernig present, Babinski absent. Spinal fluid drawn, vucin injected.

Nov. 23. Comatose; marked neck rigidity; Kernig and Babinski present.

Nov. 24. 3 a. m., rapid feeble pulse. Cheyne-Stokes respiration; died 3 :40 a. m.

Case 6 (Courtesy Drs. Beck and Pollock).—May 18, 1923. D. B., female, age 13. Present complaint, headaches; mastoid operation right.

History: Simple mastoid operation with good healing five years ago. April 21, this year, patient was taken ill with influenza, pain and discharge from right ear following. Saw local doctor, who sent her to Dr. Bradfield April 24. He found acute otitis media and mastoiditis. He operated upon right mastoid April 25; condition improved until May 6, when patient complained of severe headache; temperature went to 103 next day, right cornea became sensitive and patient complained of diplopia on looking to right.

Wound reopened May 8; condition improved until May 14, when headaches and diplopia reappeared, with some edema right optic disc. Dr. Bradfield states some vomiting but not projectile. Spinal puncture made; 16,000 cells, but normal pressure. Mother states that child was chilly and very excitable; thinks serum was injected into spine. Temperature since May 14, 103. Headaches were in left parietal region and above left eye. Patient admitted to North Chicago Hospital May 18, 1923.

Examination: Patient extremely irritable; temperature 103 rectally; pulse 108; evidence of mucopurulent nasal discharge;

tongue slightly coated; teeth negative; tonsils absent, throat negative; pupils slightly contracted, react; unable to see into fundus; ears not examined at this time; mastoid dressing on right; no rigidity of neck; no Kernig, no Babinski; a heart murmur present; lungs not examined.   Blood: Hemoglobin 75 per cent; leucocytes (unable to obtain); differential neutrophiles, 80 per cent; small mononuclears, 18 per cent; large mononuclears, 2 per cent. Urine negative for albumin and sugar.

X-ray examination: Right mastoid shows some evidence of cells in region of tegmen; middle ear apparently clear. Mastoid cavity shows previously exenterated cells and possibly exposure over sinus region. Left mastoid shows small, well pneumaticized process.

Examination by Dr. J. C. Beck: Eyes, pupils react to light and accommodation; movement in all directions normal. Ophthalmoscopic: Media clear, but fundus not seen on account of constant movement of eyes.   Ears: With noise apparatus left patient hears loud conversation.   No spontaneous nystagmus. Reflexes: Some indication of Babinski right, none left; no patellar response; Kernig negative; Brudzinski negative; lungs negative.   Heart: Sharp second pulmonic sound. No tenderness is elicited at point complained of in left temporoparietal region.

Right ear: Drain removed, is moist and has an odor; mastoid cavity gauze is soaked with exudate; cavity looks fairly healthy; there is a tract leading downward and backward to region of sinus, but nothing escaping from sinus.   Ear is radically operated; at the tegmen attici is a yellow spot, at which a little yellow material reappears after wiping.   Observation of cavity an hour later shows no new discharge. Patient under observation, without packing; ice cap to head if headache returns, aspirin; liquids.

May 19. Under ether old wound reopened by Dr. Beck: tissues look healthy; radical cavity present; an era of exposed dura is seen; burred away existing cells over sinus and tip, and curetted into several that had not been opened. The sinus was exposed for one-half inch and pricked open and free bleeding ensued. The dural opening enlarged and subdural explora-

tion made by spatula; no signs of necrosis or pus. After packing wound was sewed up and patient returned to bed in good condition.

May 20. Poor night; very restless. Spinal puncture under gas; fluid under pressure, contains 1,230 cells per cm. Patient relieved of pain and restlessness.

May 22. Not so restless; temperature 102, pulse 98; muttering and picking at bed clothes. Spinal puncture (Dr. Lewy); withdrawal of 40 cc. fluid and injection of 10 cc. vucin 1/500 into spinal canal.

May 22. General condition worse. Progressively worse to May 28, when she died.

Diagnosis: Suppurative meningitis following mastoiditis. Streptococcus infection.

Case 7 (Courtesy of Dr. C. F. Yerger).—B. A., female, age 24; admitted to County Hospital May 18, 1923. History: Headache one week, vomiting two weeks; mastoid operation right about a year ago. Earache left three weeks ago; two paracenteses, following which discharge.

Present condition: Tenderness left mastoid; perforation posterior, from which small amount serous exudation. Slight stiffness of neck; no Kernig or Brudzinski. Reflexes all active; spinal fluid under pressure, cell count 94; Pandy positive; eyegrounds negative. Diagnosis: Acute mastoiditis with meningeal irritation.

May 19. Eyegrounds reveal bilateral choked disc; slow pulse; loss of memory for names (of objects?). Diagnosis by Dr. Boot: Cerebral abscess, suprategmental, left side.

Operation May 19: Mastoid exenteration; mastoid diploetic, antrum only cell present, contained mucopus and surrounded by softened bone; cavum tympani contained cholesteatomatous material, granulations and pus; tegmen antri and tympani necrotic and were removed by curette; dura thickened, brain pulsation absent; exposed area 3½ by 1½ cm. Gifford's searcher inserted to depth of 1½ inches and liberated fetid purulent fluid mixed with necrotic brain substance. Drainage tube inserted into abscess cavity, which seemed quite large; bone cavity packed with iodoform gauze; radical operation without the plastic done.

May 21.   Examination Drs. Krumholz and Yerger: No stiff neck; no hemianopsia; reflexes all normal, pulse 88; left pupil twice as large as right and does not react as well; internal rotation left eyeball not complete; slight drooping left eyelid; horizontal nystagmus right on looking to extreme right; marked tache cerebrale; reads, tells time, talks intelligently and reasons well; sensorium not affected; Kernig absent.

May 22.   Changed mastoid drains.

May 24.   Temperature normal; feels good.

May 25.   Tube not draining. well.

May 28.   Not much discharge; skin looks inflamed.

May 28.   Fundus examination (Dr. Suker): Slight optic neuritis right.   Left: Receding optic neuritis with evidence of considerable peripapillar exudate.

May 29.   Complains of pain in mastoid region; rubber tube removed; gauze inserted.

June 1, 3.   Complains pain left ear; temperature remains normal.

June 7.   Patient wants to get up; mind active and bright; memory good for past events.

June 9, 11.   Morning headaches; vomiting stopped; small amount of pus in wound each morning.   Sits up.

June 13.   Gastrointestinal disturbance; tenderness epigastrium, disappeared after emesis.

June 14.   Dizzy; headache; vomits after eating or drinking; spontaneous nystagmus on extreme abduction more marked to right; coordination tests upper extremities normal.

June 15.   Wound probed and about 1½ ounces pus escaped.

June 21.   Complains of some loss of memory.

June 24.   Walking about.

June 26.   Forgets names of familiar people.

June 28.   Fundi normal.

June 30.   Temporal periostitis still present, with swelling. Marked contraction of color and form fields, especially left, where field averages within ten degrees.

July 2.   Has motor paraphasia (Dr. Krumholz).

July 9.   Swelling soft tissues still present.   Under local anesthesia mastoid wound opened to upper margin, exuberant

granulations curetted and drain inserted under temporal muscle.

July 16. Temperature fluctuating; had a chill.

July 17. Temperature over 100; frontal headache; wound draining profusely; opening to abscess cavity very small; catheter inserted but will not enter cavity.

July 20. Drowsiness; can be aroused; headache, vomiting; slight optic neuritis left; slight rigidity of neck; no Kernig; tache cerebrale marked; no aphasia. Spinal fluid not under pressure, cells 21,400, 95 per cent neutrophiles; Pandy and Ross-Jones positive; 15 cc. 1/750 vucin solution injected after withdrawal of 20 cc. spinal fluid. Patient died at 7:15 p. m.

Laboratory reports: May 18, spinal fluid Wassermann 3 plus; July 20, spinal fluid streptococcus hemolyticus; brain abscess streptococcus hemolyticus.

Anatomic diagnosis: Acute diffuse suppurative leptomeningitis; abscess left temporal lobe.

Case 8 (Courtesy of Dr. C. F. Yerger).—R. C., female, age 9, admitted to County Hospital June 16, 1923, with a diagnosis of convalescent scarlet fever, double suppurative otitis media, mastoiditis with subperiosteal abscess left; right facial paralysis of peripheral type. Crusting about left corner of mouth; moderate cervical adenopathy. Previous history of measles and whooping cough.

She had been given convalescent scarlet fever serum and diphtheria antitoxin. Reflexes, chest, abdomen and extremities negative, except as above noted. Watch test for hearing unsatisfactory, but it is heard in both ears at about 15 cm. Blood: Hemoglobin 85 per cent, whites 7,600, polys 59 per cent, small monos. 30 per cent, large monos. 9 per cent, transitional 2 per cent, reds 3,940,000. Temperature 101.4; pulse 120; respirations 24 on admission.

Operation, June 22, Dr. C. F. Yerger: Subperiosteal abscess left incised and drained; mastoid cells exenterated; two iodoform drains.

June 23. Temperature at noon 104.4. Culture from wound contaminated with hay bacillus.

June 24. Temperature remains high; child somewhat irrational; lower stitch removed. Urine shows trace of albumin, a few pus cells, r. b. c. and epithelial cells; lungs are clear.

Blood count: Hemoglobin 70 per cent, white cells 12,500, 79 per cent neutrophiles. "Child does not look sick."

June 25. No change. Spinal fluid under pressure; 240 cells per cm., mostly polynuclears; Ross-Jones and Pandy positive.

June 26. Markedly sick child, apparently toxic. Temperature 104; complete facial paralysis right, lower neurone type; Kernig and Brudzinski positive; moderate cervical rigidity; Babinski and Oppenheim left.

Examination by Dr. Yerger: Spontaneous nystagmus to left when looking left; does not cooperate when asked to look right, up or down; no reaction to water 68° 5 minutes on right side. Probably a lesion of labyrinth right side.

Operation: Previous to operation 25 cc. spinal fluid taken; under pressure; 10 cc. vucin 1/750 injected intraspinally; 37 cells, polys., Pandy and Ross-Jones positive; no growth from culture in 18 hours.

Left mastoid reexposed; no pathology found; right mastoid exenterated; contained pus and black necrotic material resembling blood clot. Smear shows numerous small gram positive cocci; some small capsulated diplococci and two short chains of cocci.

June 27. Child appears much better. Temperature 101.4. pulse 114. Enunciates more clearly. Another spinal puncture and 10 cc. 1/750 vucin injected. Fluid looked milky like the vucin solution; Pandy and Ross-Jones again positive, but the vucin solution gives the globulin reaction also; 250 cells per cm. polys. A pellicle formed in the spinal fluid at the end of a half hour. Smear of pellicle showed no organisms. Sterile on culture.

June 28. Marked improved; enunciates better and is able to read her letter. Kernig still positive and suggestion of a Brudzinski. Fundi oculorum negative.

General condition improved steadily since July 7; a little difficulty with wound margins; then child complained of severe headache.

July 8. Child apathetic, but answers intelligently when aroused; Kernig and Brudzinski positive; right facial palsy still marked; right fundus shows slight blurring of disc and slight engorgement of veins. Examined by Dr. Yerger, who

finds that child cannot name common objects shown her. Spinal puncture; fluid under markedly diminished pressure, 650 cells per cm., 73 per cent polys. Diagnosis: Left temporo-sphenoidal lobe cerebral abscess.

Operation, Dr. C. F. Yerger: Reopening of previous left incision with Gifford searcher; abscess is found in temporo-sphenoidal lobe about three-fourths of an inch from dural surface; about 2 drams thick yellow pus escapes; rubber tube drain; iodoform gauze packing.

July 9. Patient much improved; free discharge both ears and tube.

July 10. Tube draining well; no aphasia observed by Dr. Yerger.

July 11. A little aphasia again present; patient was allowed to go out in yard in wheel chair. From then on progressive improvement. Patient discharged August 28.

Since writing the above I have injected an acidulated Ringer solution, one minim 10 per cent hydrochloric acid to 50 cc. Ringer solution containing vucin 1/500, into the cisterna magna of a cat. Immediately on coming out of the anesthetic the cat showed a marked nystagmus of head and eyes. Head and eyes deviated to left, swift component to the right, with vomiting. The duration of this phenomenon was about ten minutes, and the cat made a good recovery and remained well for several weeks that it was under observation. As this was a one sided nystagmus trauma must be considered.

Also a man who developed pneumococcus meningitis following mastoid operation had 10 cc. of 1/500 vucin in 4 per cent salt solution injected subdurally in the mastoid region with no apparent toxic effect, but died several days later of the meningitis.

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110 NORTH WABASH AVENUE.

AMYLOID DEGENERATIONS OF THE UPPER AIR PASSAGES.

By H. E. Thompson, M. D.,

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Krakow: Amyloid contains chondroitin, sulphuric acid plus a protein similar to nucleoprotein, which is a compound of nucleic acid and protein. This is generally accepted, but Hanssen analyzed pure amyloid from a sago spleen and found no acid, though the spleen as a whole contained an excess of sulphate. Mayeda, likewise, could find no acid.

Chondroitin sulphuric acid plus water = Chondroitin plus H_2SO_4.

$C_{18} H_{27} NSO_{17}$ plus $H_2O = C_{18} H_{27} NO_{14} H_2 SO_4$.
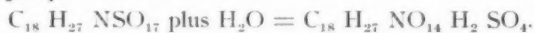
The acid is the characteristic component of cartilage and is found in the wall of the aorta and other elastic structures, uterine fibromata and bone.

Krakow gives the composition of amyloid as follows:

C = 49-50%	S = 2.65-2.9%
H = 6.65-7%	P in traces only.
N = 13.8-14%	

Neuberg found a similar composition. Neuberg also showed that amyloid contains a higher percentage of diamino-nitrogen as compared with most proteins. Apparently the amyloid in different organs varies somewhat in composition. Amyloid resembles the nucleoproteins. Neuberg considers that amyloid is a transformation product of the tissue protein similar to the transformation of simple proteins to protamins which occurs in the testicle of spawning salmon. Krakow believes amyloid differs from normal chondroitin-sulphuric acid compounds, such as cartilage, in that in the latter the union between acid and protein is a loose combination, while in the former it is a firm one, possibly being of the nature of an ester. In any event, amyloid is not essentially a pathologic

product, but a slightly modified normal constituent of the body. The exact chemical nature is as yet undetermined.

The origin of amyloid is not entirely cleared up.

The fact that chondroitin-sulphuric acid is a characteristic constituent suggests that this body may be liberated in considerable amount during the destructive processes to which amyloidiasis is secondary. This idea is supported by the fact that amyloidosis occurs particularly after chronic suppuration in bone and lungs, both of which tissues, according to Krakow, contain chondroitin-sulphuric acid.

Oddi and Kettner fed and injected rabbits with sodium salt of chondroitin-sulphuric acid without producing amyloid.

Wells' experiments, similar in character, were unsuccessful. Amyloidiasis can be caused by prolonged suppuration in chickens and rabbits or by producing chronic intoxication with bacterial filtrates. It is also encountered in horses used for the production of diphtheria antitoxin (diphtheria toxin injected).

Leupold advances the following hypothesis: In chronic suppuration a soluble protein circulates in the blood, which stimulates the formation of "defensive ferments." This protein under certain conditions is deposited in certain organs where large amounts of sulphuric acid occur. For the development of amyloid three factors are necessary: A preformed protein, an increased amount of conjugated sulphuric acid and an inefficiency of the amyloid-filled organ to eliminate the increased amount of conjugated sulphuric acid.

Most experimental work has resulted in failure (one-third successful, Davidsohn). Davidsohn failed to get it after extirpation of the spleen, and be believes the spleen produces an enzyme which causes precipitation of amyloid in tissues from a soluble precursor brought in the blood from the site of cell destructions. It is generally thought that the amyloid material is infiltrated in the form of a soluble modification or precursor and is not manufactured in the organ where found. The precursor (it has been suggested that hyalin is a precursor of amyloid, and it differs only in staining reaction) is probably brought in solution, though possibly is brought in the leucocytes.

Pollitzer states that in various infections, especially those due to the cocci, chondroitin-sulphuric acid is eliminated in the

urine, and if this is correct it has undoubted bearing on the genesis of amyloidiasis. Amyloidiasis is produced by the most varied species of bacteria and by their toxins.

Local amyloid accumulations are of some interest in considering the genesis of the usual generalized form. They occur particularly as small tumors in the larynx, bronchi, nasal septum and eyelids; as all these tissues are normally rich in chondroitin-sulphuric acid, it seems probable that the amyloid arises from a local overproduction of chondroitin-sulphuric acid, which becomes bound with proteins in situ. This makes it seem more probable that, in spite of the lack of positive experimental evidence, general amyloidiasis is due to liberation of excessive quantities of chondroitin-sulphuric acid in the sites of tissue destruction.

Another form of local amyloid is seen, particularly in the regional lymph glands of suppurating areas—e. g., the lumbar glands in vertebral caries, the axillary gland in shoulder joint suppuration. This local amyloidiasis is undoubtedly due simply to the fact that these glands receive first, and in largest amounts, the cause, whatever it may be, of the amyloid production. Less readily explained are cases of extensive amyloidiasis limited to the heart.

Corpora amylacea of prostate at times give the reactions of amyloid but are probably not true amyloid. Corpora amylacea of lungs quite often do.

STAINING REACTIONS.

1. Amyloid is stained mahogany brown with iodin (glycogen the same).

It stains lightly with Gram-iodin solution and when transferred to H_2SO_4 the color changes from red, through violet to blue, but sometimes only to brown.

2. REACTION WITH METHYL VIOLET.

1. Stain frozen sections of fresh or of formaldehyde or alcohol fixed tissue in 1 per cent methyl violet from three to five minutes.

2. Wash in 1 per cent acetic aqueous solution.

3. Wash thoroughly in water to remove acid.

4. Examine in glycerin.

3. REACTION WITH IODIN GREEN.

1. Stain fresh or hardened sections in a ⅓ per cent aqueous solution of iodin-green for 24 hours.

2. Wash in water.

3. Mount in water or glycerin. Amyloid, a violet-red; tissue, green. Stilling claims that the reaction is surer than with methyl violet.

4. Hematoxylin and eosin (Dr. Broders).

5. Crystal violet (Dr. Broders).

I am reporting the following case of diffuse subepithelial amyloid infiltration of the upper air passages for the following reasons:

First—The condition is rare. New, of the Mayo Clinic, in 1919 reported that at that time only 47 cases had been reported in the literature, and most of these were from the foreign literature. New himself reported four cases. Furthermore, only a small percentage of these 47 are of the diffuse subepithelial type, being mostly those of a more limited tumor formation.

Second—The condition in this case involved more structures than in any of the cases reported.

Third—The very great difference in opinion of various well known pathologists as to the microscope pathology.

Fourth—Apparently amyloid may be overlooked if only the usual classical staining reactions are used.

Fifth—The difficulty of making a clinical diagnosis. This case was seen by two well known laryngologists, who would not give an opinion from clinical observation.

The patient was a white American, female, age 32, in perfect general physical condition. The patient's complaint was inability to speak above a whisper, unless by great exertion, when she could produce a high pitched sound. At the age of two she had the measles, and the patient's mother told her that it was after this that her voice changed. Her voice became gradually worse until she was seven. There has been no change since. Her parents were born in Germany, but came to this country as children. Her father died of kidney trouble at the age of 55. Her mother is alive and well, except that she has double cataract. The patient is married and she has

a healthy boy, age ten. General examination of this boy, including Wassermann, is negative. His upper respiratory tract is normal. Her husband is well. Besides measles at the age of two, she has had scarlet fever and whooping cough. There have been three miscarriages.

The following is the report of the professor of medicine of a State University. The following is Dr. ———'s report on Mrs.———'s physical examination: "Is entirely negative, except for the scarring in the throat and an accentuated aortic second with slight enlargement of the heart. She gives a history of three miscarriages, but we understand that the Wassermann done by the State Board on July 15 is negative. In spite of the negative Wassermann, we would make a diagnosis of tertiary syphilis in view of the appearance of the throat and the accentuated aortic second. There is probably a syphilitic aortitis." We have had, during the past four years, repeated Wassermanns made, and they have been uniformly negative; also spinal fluid was negative. I believe we can say that she has not syphilis, in spite of the report of the clinician, by reason of the reported negative Wassermann and negative spinal fluid, healthy child with negative Wassermann and no signs of clinical syphilis except a suspected syphilitic aortitis. The total white count and differential are normal. The mucous membrane of the vagina and rectum are normal.

Examination of the upper respiratory tract: The parts involved are as follows: Nasal septum, some involvement anteriorly and posteriorily on both sides; turbinates are normal; the posterior pharyngeal wall, tonsils, anterior and posterior pillars, soft palate, uvula, base of tongue, epiglottis, true cords, interarytenoid space and the entire circumference of the subglottic space—also the upper end of the trachea. The hard palate is not involved to the extent of the other parts. There is a diffuse infiltration of these parts involved, giving the mucous membrane the appearance of pebble leather. The mucous membranes are yellowish gray, and in certain parts, especially the pillars of the tonsils and the soft palate, the infiltration is so general that no distinct nodules can be made out, one nodule merging into another. In other places, especially over the hard palate, distinct discrete nodules can be seen. Nodules vary in size, some 2 to 6 or 8 mm. The tissues are hard and unyield-

ing to the touch and are slightly anesthetic. One pathologist, seeing the case clinically, said the appearance suggested to him the appearance of a typical sago spleen. The vocal cords are generally thickened, the edges are rough and the infiltration of the interarytenoid space prevents anything like proper examination. There is also involvement of the margins of both lower lids, on the left side the punctum being blocked."

Previous to the appearance of New's article in the Laryngoscope in 1919, I had sent pieces of tissue to three well known pathologists and received the following reports: First, the tissue consists of two layers of stratified squamous epithelium which converge towards each other at one end of the section, but are separated by a wide break in the tissue. Between the layers of epithelium there is a wide layer of connective tissue. The epithelium is not horny around the surface. The structure does not contain any evidence of tonsillar tissue, but may be a polyp from the pharynx. No lesion here is definite enough to permit a positive diagnosis. But in the connective tissue part of the section the blood vessels show a considerable degree of obliterative endarteritis. At one corner there is an area of hyalin degeneration, around part of which there are small round and epithelioid cells. The lesion suggests a syphilitic process more than anything else. A definite opinion, however, cannot be based on the specimen.

Second—Gross description: Four small pieces of tissue taken from thickened vocal cord, posterior pillar of pharynx, posterior wall of the pharynx and interarytenoid nodule. Microscopic description: Samples from one, two and three all show the same increase of connective tissue in the mucosa and submucosa. The blood vessels are increased in number and have exceedingly thick walls. All the coats are involved in this thickening, especially the middle coat. Most marked is the connective tissue about the mucous glands. The acini are few in number and are surrounded by broad bands of connective tissue which has undergone some hyalin degeneration. The squamous epithelium covering the tissue is hypertrophied. About many of the blood vessels is a very marked round cell infiltration.

The iodin and methyl violet stains used for detecting amyloid were invariably negative.

Third—Sections show much thickening of the epithelium, the surface of which is hornified. The tissue underneath is uniformly dense, almost hyalin, without any differentiation and with only an occasional nucleus. No tubercles, no giant cells: the few vessels there are have thick walls, and only in one or two places are there indications of slight leucocytic infiltration. I am unable to make any diagnosis, but do not these appearances point to rhinoscleroma?

As you will see from these reports, we are not able to make a definite diagnosis. After reading New's article I believed this case to be similar to the ones he reported as diffuse subepithelial amyloid infiltration. I then sent a piece of tissue to him with the history of the case. He turned the tissue over to Dr. Broders, who without any knowledge of the case made a diagnosis of amyloid, using hematoxylin and eosin for stains.

Amyloidosis of the upper respiratory tract occurs as part of a general amyloidosis or as a local amyloidosis. New classifies the latter under three types:

1. Diffuse subepithelial infiltration.
2. Tumor forming local amyloidosis.
3. Amyloid degeneration of a preexisting tumor.

This case is unique in that, first, the condition is not part of a general amyloidosis but is entirely a local condition. Second, the condition involves more structures than in any previously reported case. Third, the condition began at a very early age.

In working on this case early, before the real diagnosis was made, we injected some of the tissue into the peritoneal cavity of rabbits and also into the testicles. At the postmortem of these animals there was nothing found grossly or microscopically to help us in a diagnosis.

At one time she was given old tuberculin and gave a positive general reaction, but no local reaction in the throat. Stained specimens or cultures never revealed any but ordinary organisms such as we find in the upper respiratory tract. The treatment was to remove the rough edges of the cord and some of the infiltration from the interarytenoid space, using a Jackson speculum. This was done four years ago, and her voice was improved and has stayed improved since.

Why this condition should occur I cannot say, but I present this as a suggestion: Local amyloidosis occurs more frequently in the upper respiratory tract than elsewhere, and for this reason I believe we must look for some local chemical constituent of these parts that under certain pathologic stimuli will produce amyloid.

In the first part of this article we said that chondroitin-sulphuric acid is normally abundant in the tissues of the upper respiratory tract and also that amyloid is probably the product of the chondroitin-sulphuric acid on the normal protein of the tissue. Thus any inflammatory reaction in the tissues of the upper respiratory tract that would stimulate the production of an already abundant amount of chondroitin-sulphuric acid would provide for the increase of this acid which is necessary in the formation of amyloid.

We have normally the preformed protein. The inflammatory reaction gives rise to an increase of chondroitin-sulphuric acid. We have also to presuppose that in these cases where local amyloidosis occurs that there is an inefficiency of the amyloid filled organ to eliminate the increased amount of conjugated sulphuric acid.

XVII.

LARYNGEAL EPILEPSY.

By Beaman Douglass, M. D.,

New York.

By this term I mean a distinct epileptiform attack of the more severe variety of the "petit mal" group of symptoms produced by a laryngeal irritation. Such cases are sufficiently rare to be interesting, and I hope interesting enough to excite discussion. A review of the literature shows reports of many cases and none reported since 1898.

Since Charcot, the famous French neurologist, first described this malady and gave it a name there have been but relatively few cases reported. In all, I am able to find about 95 case reports, scattered through the literature up to 1896. Since then, probably on account of the greater rhinologic interest, the literature is void of these cases.

Charcot clearly distinguished between glottic closure or spasmodic laryngitis, causing attacks such as we see in pertussis, and this condition of laryngeal epilepsy, in which there is entire muscular relaxation with unconsciousness but no spasm of the glottis.

Writers since his time have not been so careful, and the reports show several cases of spasmodic laryngitis classed as laryngeal epilepsy.

There has been also a confusion in the terms used to name the condition. Some call it laryngeal vertigo, others laryngeal spasms and still others laryngeal epilepsy. The attacks described are all mostly of the "petit mal" group, although some writers describe a full fledged convulsion of epilepsy. These were, however, mostly in cases with a bad epileptic antecedent family history. The cases were mostly males of middle life, usually nervous men with too much fat and blood.

Recovery is the usual result. Only one fatal case is reported, and this by Schaedewaldt (Arch. für Laryngologie und Rhinologie, 1896, 246), where the patient fell unconscious

during an attack of coughing and died immediately. As no autopsy was made, this case is open to doubt.

Knight, F. J. (New York M. J., XLIV—1886), contributes a valuable paper on this subject. His cases are clean cut and well described.

McBride, cited by Phillips (Trans. New York Acad. of Med., 1892, p. 61), evidently is confused on the subject, and describes cases of spasm of the glottis as epilepsy. Phillips draws attention to this and reports a very well described case of the real laryngeal epilepsy.

Kercher (J. Am. M. Ass., 1893, 604) says it is a neurosis which is cured by treatment of the respiratory tract. His case reported is evidently spasm of the glottis, since the epileptic elements are missing.

Mulhall (Laryngoscope, 1898, 167) wanders for an explanation of his case into the well worn and popular road of uric acid in the blood. The description of the attack is, however, very clear and convincing.

By far the best paper was contributed by Getschell (Boston M. & S. J., 1896, 486). This paper is a valuable and interesting composition and contains a bibliography covering 77 case reports. He concludes that a paroxysm of coughing which always produces congestion of the cerebral vessels may cause syncope but not unless there be an existing disorder of the central nervous organ.

Textbooks on nerve diseases are silent on this subject. Some of the older works on laryngology mention epilepsy from respiratory irritation. A glance through the literature of epilepsy shows conclusively that a peripheral irritation may cause epileptiform attacks, and among the established causes are found gastric conditions, the sight of a cadaver, nasal disease, hypertrophied tonsils, tooth pain, uterine stenosis, eye strain, elongated prepuce and fibroma of the hand; certainly a varied enough list to establish the causative factor of the attack as a peripheral irritation.

The following case came under my observation this winter (1923) :'

Mr. H., aged 59—always healthy except for attacks of laryngitis, usually coming on with the winter months. After a few weeks of coughing and hoarseness the attack would wear away, to be followed a year later by another similar one.

The patient was one of those possessing marked sensitiveness of the mucous membrane, which rendered all local examination and treatment very difficult. It was necessary at each treatment to completely cocainize the pharynx and larynx in order to make an examination and to give treatment.

In September, 1922, he appeared in my office from his country place and stated that he had suffered all summer from a troublesome cough and hoarseness. The cough, which was spasmodic in character, was worse at night, and sufficiently active to keep him awake.

An examination after cocainization showed a marked subacute pharyngitis and laryngitis. Heart and lungs normal, blood pressure 140, urinalysis normal and all other organs normal.

Subsequently a series of X-ray pictures confirmed these findings in the chest.

The coughing attacks were at first controlled fairly well by local treatment and small doses of heroin, but the patient did not make the usual recovery. The epiglottis seemed to be very much inflamed, was boggy, velvety and very red, and did not improve like the rest of the larynx and the pharynx. This thickened and inflamed epiglottis resisted all treatment and evidently was the cause of the coughing spells, which were frequent and severe.

Early in December the patient reported that he had fallen unconscious during a coughing spell—a fact which excited his family and himself very much. He was told to stay in his room, and now for the first time I had an opportunity to observe the "spells."

His cough was certainly spasmodic and severe, but there was never any larynx spasm or obstruction to respiration. The "spells" were of two kinds; sometimes a coughing attack would be established, and after some pharyngeal choking and gagging the patient would succeed in expectorating a mouthful of mucus and the "spells" would cease. At other times a "spell" would start in the same way with coughing and gagging, but there would be no expectoration; the attack would continue, and suddenly the patient's face would become very red from vasomotor dilatation, the eyelids would open wide, the eyes roll upward, the mouth close tightly. The cough

would cease, the tongue protrude, and if caught between the teeth would bleed, and some froth would always appear on the lips. The head would fall forward from relaxation of the posterior neck muscles, and the patient would fall completely unconscious for a few seconds.

On recovery from the unconsciousness there was mental confusion for a few seconds more, after which the patient was normal again.

It was a curious fact that these epileptiform attacks never came on if the patient could expectorate.

On some days there were as many as six attacks—on other days not so many. After the patient had bruised himself badly by falling during these attacks he consented to stay abed and, later on, learned to lie down as soon as an attack of coughing would start.

All my pet antispasmodics failed to relieve the case; chloral, bromids, heroin and codein were of no use. As soon as the epileptiform character of the attack was noticed I succeeded in controlling the attack with luminal and antipyrin. With increasing doses of these remedies the epileptiform attacks disappeared, and continuance of local throat treatment helped the epiglottis. The patient finally departed for a warm climate, from which he writes me that his attacks have gradually disappeared and that now he is entirely free from both the attacks and the cough.

CONCLUSION.

1. Laryngeal epilepsy is a state of unconsciousness produced by an attack epileptiform in character, originating from a laryngeal irritation, usually from some form of laryngitis.

2. It must be differentiated from pertussis, the differentiation being that in laryngeal epilepsy there are an absence of embarrassment to respiration by laryngeal spasm and the presence of a temporary muscular unconsciousness of short duration.

3. Cases are mostly in males of middle age who have over-indulged in the good things of life.

4. An epileptic taint is usually absent.

5. Recovery is prompt after laryngeal irritation subsides.

XVIII.

HEARING IN THE PRESENCE OF A NOISE.

By Frederick W. Kranz, Ph. D.,

RIVERBANK LABORATORIES,

Geneva, Ill.

It is the testimony of many deaf persons that they "can hear better in a noise," this noise being the noise of a street car, an elevated train, a machine shop or some similar type of disturbance. The observations which have been actually made seem to be on the fact that these persons can carry on a conversation with a normal hearing person more easily in a noise than otherwise.

A number of tests have been made on this type of deafness. The tests were made by talking to the subject in a monotone, repeating over and over some phrase, and either (1) using a telephone receiver which was emitting a loud tone of 120 cycles per second and alternately placing it near to his ear (one to three inches) and taking it away, or (2) using a specially designed bone conduction type of telephone receiver actuated by a 120 cycle current and alternately placing this on his head and taking it off, there being, sufficient intensity to give the subject a loud subjective sound when on his head. The presence of the disturbance caused by either the bone or air conducted sound did not in any case result in any improvement in the subject's ability to hear or to understand the phrases being spoken.

It seems that the determining factor in the ability of some deaf persons to converse better in a noise than otherwise is undoubtedly the increased loudness with which a normal person talks in the presence of a noise. The normal person, of course, tries to talk so that the sound of his voice will predominate over the disturbing noise as judged by his own hearing. The actual ratio of the objective intensities of the speech and the disturbing noise striking the ear is the same for the normal and the deaf person, but due to the difference in the acuity of the two persons the speech sound will for the deaf

person be much farther above the low limit of audition as com-
pared with the disturbing noise than will be the case for the
normal person.   This point may be illustrated with numbers
to represent the intensities.   Let the intensity of the disturb-
ing noise be represented by 6, and that of the speech of the
normal person in the presence of the noise by 8.   Let us say
that the normal person can hear any sound with an intensity
greater than 2, and that the deaf person can hear any sound
with an intensity greater than 5.   For the normal person, the
speech and the noise are 6 and 4 units of intensity, respect-
ively, above his limit of audition, while they are correspond-
ingly 3 and 1 for the deaf person.   The latter would be at an
advantage under these conditions.   Also another factor, not
taken into account in the above consideration, probably enters
into the case.   This is the difference in frequency between the
sounds concerned in speech and those of the noise.   The latter
are probably of much lower frequency than the former, and
also it is a fact that people with middle ear trouble, who are
the ones who report the phenomenon under discussion, are
particularly deaf at the lower frequencies.   So for these people
there is probably a much wider difference between the sub-
jective intensities of speech and the usual disturbing sounds
than is the case for the normal hearing person.   The noise
bothers the deaf person less than it does the normal person.

This undoubtedly constitutes a sufficient explanation of the
observation that some deaf persons are better able to converse
with a normal hearing person in the presence of a noise than
otherwise.   A more extended series of tests on this type of
deafness is planned.   One interesting experiment suggested
by the above can be made with the aid of two deaf people, each
of whom finds that he can converse with a normal person more
easily in a noise.   The above proposed explanation would indi-
cate that if their deficiencies in hearing were similar, these two
people in conversing with each other would not find the noise
to be of any advantage.

XIX.

TREATMENT OF POSTOPERATIVE CAVITIES OF THE MASTOID PROCESS WITH RUBBER BALLOONS.

By Prof. B. Torok,

Budapest, Hungary.

Experiments with rubber balloons instead of packing in the treatment of wounds following radical mastoid operations were begun in 1913. These experiments proved to be rather encouraging, but the outbreak of the World War interrupted the work, owing to the impossibility of obtaining proper material for the balloons. These balloons are thin walled, soft and pliable, so that when inflated in a cavity they conform perfectly to its shape and adhere everywhere to its walls. When inflated the balloons enter into·all the corners, filling them entirely. They are manufactured in three sizes; the medium sized one is $2\frac{1}{2}$ cm. by $1\frac{1}{2}$ cm., and is attached to a 5 mm. rubber tubing which is 10 cm. long.

So far in eighty cases balloons have been used exclusively; in no case was packing resorted to.

The long, tedious and extremely painful after treatment following a radical mastoid operation is a very important chapter in otology. The performance of this work takes a great deal of time and energy of the attending physician as well as that of the patient. It is, therefore, of prime importance to simplify and shorten this period in an operation, thus often preventing serious and sometimes fatal complications. How often do we meet patients who, when advised to be operated on, refuse because they cannot spare the time required and later return with serious complications? It is easy to understand that a man who has a family to support will hesitate to undergo such an operation because the after treatment lasts for several weeks; especially will he hesitate when this long standing chronic disease does not cause him, momentarily, inconvenience.

Reviewing the process of healing which takes place in the cavity, we find first of all that the walls of the cavity become covered with granulation tissue. This is the first stage; in the second stage the granulation tissue contracts and turns into connective tissue. At the same time the whole surface is covered by epithelium, which slowly creeps from the wound edges forward until it covers the entire surface. The process of healing in these two stages varies considerably; the patient's general condition, his age, the microorganisms which have caused the infection must all be taken into account, as they may greatly protract the time of recovery. The healing process may also be retarded if, during the operation, all the diseased tissue has not been removed, or the cavity not carefully cleansed, or if bone particles, accidentally left in the cavity, later act as irritating foreign bodies.

The mode of after treatment will also influence the time required for the completion of the healing. Neither packing with gauze nor treating the cavity without packing is satisfactory. The former is very painful, and it often happens that fine threads of gauze, which are easily overlooked in the cavity filled with bleeding granulation tissue and secretion, are left behind. These acting as irritating foreign bodies cause hypergranulations and hinder the growth of the epithelium. In removing the gauze the fine tender epithelium is often brushed off and the granulating surface mechanically irritated, giving rise to hypergranulations.

In treating the cavity without packing there is nothing that will check the growth of granulation tissue, and the result will be an uneven surface with many elevations and depressions.

Experience has shown us that the epithelium slowly creeps forward until it reaches an elevation or depression; either of these will stop its progress. A smooth walled cavity, void of hypergranulations, will be covered by epithelium in the shortest period of time. The gentle, moderate, easily regulated, uniform pressure obtained by the use of rubber balloons seems to be ideal for producing such a smooth walled cavity. The mechanical irritation of the gauze is eliminated by the even and smooth surface of the balloon; its insertion and removal are practically painless; there is no possibility of leaving irritating threads of gauze behind, and the result in every case

is a smooth granulation surface which is on the same level as
the plastic flaps covered with epithelium.   If later a Thiersch
plasty is done the transplanted epithelium is uniformly pressed
against the smooth surface and is not brushed off, as often
happens at the removal of gauze packing.   The insertion and
removal of the balloons are not painful; only when inserting
it does the patient occasionally register pain and this is always
due to the balloon being too tightly inflated; by letting some
of the air escape the pain in all cases can immediately be
relieved.

The procedure is as follows: After the operation the cavity
is carefully cleansed, all diseased tissue removed, special care
is taken to remove small splinters of bone from the hypo-
tympanum.   All granulation tissue is removed with the excep-
tion of those situated in the pelvis fenestræ ovalis, which re-
gion should not be touched, notwithstanding the fact that
granulations left there may retard the quick and uneventful
recovery.   Körner's plastic operation was first tried, but in
two cases perichondritis developed, which I attribute to the
pressure exerted upon the flap by the rubber balloon; at the
subsequent operations Stacke's operation was resorted to where
such complications cannot arise.   The flap is made thin so
that it should easily conform to the walls of the bony cavity,
and after sutures have been inserted the cavity is packed with
vioform gauze.   The sutures are removed on the fourth day
and the packing removed on the fifth, on which day the first
rubber balloon is inserted.

A small piece of gauze is placed loosely in the tympanum
and, by means of forceps, the sterilized folded balloon, an-
ointed with glycerin, is pushed into the cavity.   The rubber
tube of the balloon is then connected with a Record syringe
and carefully inflated until it loosely fills the entire cavity.
The rubber tube is now grasped with an artery clamp and a
bandage applied.   The balloon is removed on the next day
and the cavity cleansed with a normal saline solution or with
hydrogen peroxid.   In most cases the balloon is reintroduced;
only in cases where there is much secretion is the balloon left
out for one day.   In such cases a small gauze strip is intro-
duced into the opening of the tympanic cavity.   After having
inserted the balloon for six or eight consecutive days we find

a well formed cavity with smooth white walls and with signs
of beginning epithelialization. From now on the cavity is
left open, removing the secretion daily by gently irrigating
it with saline solution and then dusting it with boric acid or
with vioform powder. The cavity should never be cleansed
with gauze, as this will destroy and brush off the new epithe-
lium. In uncomplicated cases we have a cavity covered with
epithelium in three or four weeks.

In a series of cases I have applied Thiersch flaps on or about
the tenth day when the bony walls were already covered by
a thin layer of granulation tissue. Without exception the
flaps have taken, thereby considerably shortening the period
of epithelialization.

The good results obtained in chronic cases lead us to try this
same procedure in cases of actue mastoiditis. A number of
cases have been treated in this manner. The incision is made
exactly in the same manner as in a radical operation, and the
mastoid is cleansed of all diseased tissue. I consider this a
very important point, and disagree with Lange when he says
that in the after treatment of antrotomia retained inflammatory
focuses are beneficial in promoting the formation of granula-
tion tissue and bone tissue. In my opinion, it is a rather
dangerous way of promoting granulation tissue formation and
also superfluous in this method of after treatment, as we do
not aim to fill the cavity with granulation tissue. It is, there-
fore, important that the removal of all diseased tissue should
be carefully accomplished, not only in the direction of the
tegmen and sinus, but also in front of the antrum, around the
aditus and in the pneumatic cavities of the zygoma. Our ex-
perience has taught us that in almost all cases where a fistulous
opening remains, the fistula leading toward the vicinity of the
.antrum, at the second operation diseased focus was found in
the vicinity of the aditus.

In removing all diseased tissue, we also remove the pos-
terior wall of the auditory canal as in a radical operation, also
the small cells around the aditus, thereby exposing the pos-
terior superior corner of the tympanic cavity which, in our
experience, has no detrimental effect so far as hearing is
concerned. In removing the posterior wall of the auditory
canal great care should be taken to do it properly so that the

level of the tympanum should not be too far below it.   After
having prepared the cavity the after treatment is similar to
that of a radical operation.   It is, however, advisable to wait
with the plasty as well as with the sutures for a few days.
until the inflammatory symptoms have somewhat subsided.
This is then followed by the treatment with the balloon.

A similar operation for acute cases was recommended by
Winkler in 1904, but evidently found no followers.   I find the
procedure especially advantageous in lymphatic, anemic pa-
tients, where, as experience has taught us, the formation of
granulation tissue is very slow, the recovery protracted, lead-
ing often to deep cavities, fistulæ and a persistent retroauric-
ular opening.

This method of operating is, in my opinion, also important
from the standpoint of the socalled recurrent mastoiditis cases.
Wolff, Frey, Bondi, Lange and others have investigated the
subject and found that the cavity is not solidly filled with bone
and connective tissue, as described by Schwartze and Politzer,
but contains cysts and cavities, some of them communicating
with the tympanic cavity.   In case of an inflammation in the
tympanic cavity these cysts easily become infected, causing the
recurrent mastoiditis.   By this method of operating this com-
plication is eliminated.   We have had several cases operated
on by this method, who later developed a severe middle ear
inflammation without causing any serious complications.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday, November 5, 1923.

The President, Dr. John A. Cavanaugh, in the Chair.

Bezold's Abscess.

Dr. J. Holinger presented a case of Bezold's abscess. The patient was a man fifty-eight years old, who showed a scar behind and above the left auricle through the tip of the mastoid down the neck to within three fingers of the clavicle. One centimeter below the mastoid the scar had a branch forward and downward, and five centimeters to the rear there was another short scar as a consequence of a counter incision. These scars gave a slight idea of the large size of the abscess, which reached nearly around the whole left half of the neck. Medially from the mastoid process the typical large cell which led to the perforation into the neck was found in the floor of the temporal bone. It extended forward to close to the facial nerve, yet no injury was done to the nerve at the operation. The cell was attacked from behind the mastoid process, the dura was exposed and granulating, and at the time of presentation the drum membrane was closed and hearing was two meters for whisper.

This patient was one of four with nearly identical conditions that Dr. Holinger observed last spring. They all had been treated before he saw them from six weeks to two and one-half months and had been seen by general practitioners and surgeons. This patient was treated for several weeks in a well known clinic. The connection of the suppuration of the ear with the swelling on the neck was not even suspected. The unusual feature of all four cases was the slow course and the fact that they all got well. The pus does not always stop in the neck. The descent into the posterior or anterior mediastinum is sometimes very rapid. Several years ago Dr. Holinger observed a woman who died from acute posterior mediastinitis due to descent of the pus along the vertebral column. Death occurred within forty hours after the first symptoms of an acute otitis media.

Foreign Body in the Bronchus.

Dr. Edwin McGinnis presented three cases of foreign body in the bronchus.

Case 1.—C. H. F., age 4 years, on September 7, 1923, inhaled a glass headed shawl pin about two inches long. He strangled slightly and the mother saw the pin at the base of the tongue but was unable to remove it. X-ray revealed the pin in the right bronchus, pointing upward. The pin was removed by bronchoscopy on September 11th, some difficulty being experienced in freeing the point, which was imbedded in the mucous membrane. The temperature, which was 100.2° F. on admission to the hospital, gradually subsided and recovery was rapid.

Case 2.—A boy, aged 3 years, on June 6, 1923, apparently swallowed a nail with which he had been playing. The stools were watched for six days but the nail was not recovered. The boy developed a slight cough, and X-ray examination showed the nail in the right bronchus. No pain was complained of. On June 13, 1923, upper bronchoscopy was done, without anesthesia. During the manipulation the nail was grasped but slipped out of the forceps and went into the left bronchus. It finally was grasped and removed. The temperature was 99.2° F., and rales were present on both sides the day of operation. Recovery uneventful.

Case 3.—T. E., age 2 years, was playing with paper clips and one slipped down his throat. He strangled, became cyanotic and commenced to cough violently. X-ray examination showed the clip at rest in the upper part of the trachea. The child had a crowing respiration and spasmodic cough. At the Presbyterian Hospital, with direct laryngeal speculum the clip could be seen, was grasped with forceps and withdrawn. Recovery uneventful.

"Functional Tests of Hearing."

Dr. Robert Sonnenschein gave an illustrated address on "Functional Tests of Hearing."

Paper: "Vucin—The Use of, in Intracranial Infections."*
By Alfred Lewy, M. D.
DISCUSSION.

Dr. Robert Sonnenschein said he did not know whether the very favorable case referred to by Dr. Lewy was the one

*See page 254.

Dr. Lewy had seen with him or not. This case was that of a little girl, aged nine or ten, who had had an acute otitis media for ten days. When seen by him she had a rapid pulse, a temperature of 104° F., rigid neck, severe headache, Kernig and showed the picture of a well developed meningitis. Spinal puncture showed cloudy fluid under markedly increased pressure, 3,000 cells per millimeter, bacteria in smear but not on culture. At operation, which was performed at once, much destruction was found, with the sinus exposed and the dura quite gray. The following morning another spinal puncture was made and the cell count was 7,000 and vucin was injected. The patient had a stormy time for four or five days with a high temperature, but gradually made a complete recovery. Dr. Sonnenschein thought that as long as there was no harm done when the vucin was injected at the time of the spinal puncture there might be no objection to its use.

DR. HARRY L. POLLOCK said that about a year or so ago he had a case of meningitis in a young lady which followed a very slight otitis media. The otitis media cleared up within a couple of days, but the other symptoms developed, viz., labyrinthitis and meningitis. Spinal puncture revealed as high as 8,000 leukocytes, but culture was sterile. No bacteria were found. They cultured it and injected guinea pigs and rabbits, and at that time Dr. Lewy advised injecting vucin, but Dr. Pollock thought this was not advisable unless bacteria were present. The patient ran a stormy course for two weeks. They did several spinal punctures and used intraspinal medication of serums, and the patient recovered sufficiently to sit up when, without any warning, she got a recurrence and in the course of several weeks died. During the course of the illness twenty spinal punctures were made but no bacteria were found.

Dr. Pollock called attention to the fact that some of the cases Dr. Lewy referred to as "arrested" or "improved" and said that they found many which improved temporarily without any treatment, the socalled protective meningitis. They tried vucin treatment on another case which followed two mastoid operations two weeks previously when streptococci were found. Vucin was injected but the patient died. Dr. Pollock thought

that none of the cases were conclusive unless bacteria were found. So many of the socalled protective meningitis conditions will give a high count and a cloudy fluid but no bacteria. He believed that these were all extreme cases and that the vucin should be used as early as possible, especially when bacteria are present.

Dr. Peter Bassoe said that he knew nothing about vucin, as this was the first time he had heard of it, but it had occurred to him that an agent like this which is possibly neutralized by the spinal fluid, would be better injected by cistern puncture simultaneously with spinal puncture, thorough irrigation being thus possible. Such irrigations have been done with other substances, introducing the fluid by cistern puncture and allowing it to run out by means of the spinal puncture. He thought cistern puncture was a comparatively easy and safe procedure.

Dr. Norval H. Pierce asked for Dr. Carlson's opinion of what he was about to say. If the spinal fluid could be changed into a germicidal fluid, how much effect could be expected on tissues in cerebrospinal meningitis? Is not death produced not by the content or condition of the cerebrospinal fluid but by the pathologic process that goes on in the arachnoid and especially in the pia; in other words, within the blood vessels themselves or in the perivascular spaces? It seemed to him that no matter what antiseptic might be injected into the cerebrospinal fluid it would have very little effect upon the condition of the vascular system of the pia or arachnoid unless the injected substance was specific for the pathologic process in the tissues and gained access thereto. It is known, for instance, that the reason there has been so much late syphilis is that the arsenic preparations that have been used up to the recent past have failed in their penetrative powers. Now preparations which have greater penetrative power are being produced, so that the spirochetes can be killed off in the nervous tissue itself. Dr. Pierce believed this same thing had a bearing on the question of all injections into the cerebrospinal fluid, and asked whether the hope that these injections, whatever they may be or whatever effect they may produce in the cerebrospinal fluid per se, might be beneficial in the pathology of septic meningitis, or would they prove futile?

DR. A. J. CARLSON (by invitation) was inclined to agree with the general trend of Dr. Pierce's remarks and said that certainly he should not feel like injecting vucin into the kind of patients described until he knew something more of what the drug did when introduced simultaneously with bacteria, or with bacteria introduced a little earlier or a little later, in the spinal canal of experimental animals. He was disappointed that Dr. Lewy had not done this before using it on patients.

As to the fundamental question raised by Dr. Pierce, killing the bacteria in the spinal fluid would probably be of very little avail unless the drug introduced penetrated freely through the meninges and through the cells and connective tissue into the surrounding areas. One very clear example of that is offered in the gut. In the days when most of those present were general practitioners they had probably tried to use the socalled intestinal antiseptics. A number of years ago in some research work in their laboratories, Dr. Dragstedt attempted to sterilize the gut by all means short of killing the intestinal mucosa. He applied antiseptics in strengths that could not possibly be applied anywhere else, on a small portion of the gut, but found that the bacteria between the cells were still there and ready to do whatever damage they could in that position. However, while very little is known as to the nature and control of cell permeability, there is no doubt that many substances go through or diffuse through the tissues entirely independent of any circulation of fluid, cerebrospinal, lymph or blood. Dr. Nultzer showed a number of years ago that one could cut out the heart of a frog and inject a drug peripherally and that after a while the drug would work through the entire body by diffusion. Bactericidal drugs to be effective in meningitis must go through the membranes and to the cells. Sterilization of the spinal fluid itself means very little.

DR. ALFRED LEWY asked Dr. Carlson to state what the present status of our knowledge is regarding the diffusion of the spinal fluid.

DR. CARLSON said that our knowledge was very limited. We do not know where it comes from or where it goes to or what it does while it is there. Some claim that it is secreted by the choroid plexus, and there is some evidence in

favor of that theory. Dr. Weed at Baltimore and Dr. Cushing at Boston take this view, but it is still a theory. Dr. Becht at Northwestern has done some work that supports the theory of filtration from the blood. There seems to be no doubt that it can be absorbed along the course of the meninges. Dr. Carlson thought that if it was possible to remove the entire choroid plexus the spinal fluid would still form.

He considered the point raised by Dr. Bassoe important and would not expect much from vucin injected in small amounts and under slight pressure into the lumbar region, as he thought very little if any would get up into the brain. He believed the drug would be absorbed before it reached the brain, but if drainage was established, up or down, mechanical transport of the drug would be facilitated. However, as Dr. Pierce said, if vucin does not get into the cells or does not retain its bactericidal action after it gets into the cells direct bactericidal action in the spinal fluid itself would be of little clinical value.

DR. J. HOLINGER maintained the observations of Professor Mouckow, which seem to show that the cerebrospinal fluid is not homogeneous, at least not always. For example, it was found that one may get a positive Wassermann reaction in the fluid of the lower part of the vertebral columns and a negative Wassermann reaction in the upper part. Furthermore, in cases of injury it has been found that in one part of the spinal canal the cerebrospinal fluid may be decidedly bloody, while in another part it is clear. If there was such a free interchange of fluid from the brain down to the lumber region there would be no explanation why the whole of the fluid should not be blood tinged almost simultaneously. Of course these observations have to be confirmed by others, but to him they were very surprising.

DR. CARLSON asked if Dr. Holinger meant to imply that in a healthy individual the spinal fluid was not practically homogeneous.

DR. HOLINGER replied that so far only in pathologic conditions it had been shown. We usually do not make multiple punctures of the vertebral canal in healthy individuals.

DR. CARLSON believed there were many reasons why it should be homogeneous in the normal individual. The varia-

tions in the Wassermann reaction may be due to the location of the gumma.

DR. GEORGE W. Boot said that he could vouch for the fact that one could do a spinal puncture within an hour after a skull fracture and get bloody spinal fluid. He had done this repeatedly. It is also possible to inject the spinal canal and cure disease, as is often done in epidemic meningitis. Another point brought out by Dr. Boot was that streptococcus meningitis occasionally recovers after operation. He has had one such case. He saw a case in consultation with Dr. Holinger where a patient with streptococci in the spinal fluid recovered after a mastoid operation. He thought most of the members present had seen a few such cases. Alexander of Vienna reported such a case many years ago.

DR. A. L. TATUM (by invitation) thought it was well known that any disinfectant had to be pretty strong in order to kill a germ and was then apt to kill the tissue. The most marked successes have been such as Dr. Boot mentioned, by vaccines and serums, and the work with tryparsamid in the fourth stage of syphilis. It has been shown that this drug has no effect in the early stages but has a very marked effect in the late stages. This can hardly be understood at present, but what Dr. Tatum wished to emphasize was that one could not pour a disinfecting solution of bichlorid, for example, on a tissue and expect to conserve it in the normal state. Disinfection in vivo is quite a different thing from disinfection in vitro.

DR. LEWY (in closing) said that Dr. Bassoe's suggestion was included in his paper. He intended using vucin in the cisterna magna or by subarachnoid irrigation as soon as he was satisfied by animal experimentation that it was safe. While injection of 10 cc. of one to five hundred solution into the dorsal spine appeared to be safe, he did not know what the result might be if the same concentration was injected directly in the region of the victal centers. He had made only one such experiment so far.

Regarding Dr. Carlson's suggestion that the drug should be injected experimentally at the same time or soon after infection of the meninges in animals with controls, these experiments had all been done by Kolmer and Idzumi (quoted in the paper), using ethyl hydrocuprein hydrochlorid (opto-

chin), a closely related cinchona derivative. Vucin is said to be no more toxic but four times as germicidal. Kolmer concluded from his tests that the drug had preventive value if injected four to six hours after infection took place. With this as a basis, Dr. Lewy undertook clinical verification. Recovery from meningitis with living cultures of pneumococcus or streptococcus in the spinal fluid almost never takes place. Dr. Boot's case is a rare exception. The exceptions are so rare that we are inclined to believe that the reported cases are due to extraneous infection of the cultures or some other error in technic. If any treatment would reduce the mortality to 50 per cent or 75 per cent, he thought it would attract attention without control statistics.

Dr. Lewy had recently seen an article in the British Journal of Otology, by Dr. Davis, describing the result of injection of coloring matter under the arachnoid. Injected through the internal auditory meatus it all gravitated into the interpeduncular space only; injected in front of or behind the lateral sinus, it gravitated into the cisterna lateralis and to some extent into the cisterna magna, but in no case did it show in the spinal canal. This made Dr. Lewy wonder if injections into the spinal canal would diffuse into the basilar region where they might do some good.

In regard to Dr. Tatum and Dr. Pierce's idea that it is a fallacy to expect local applications of germicides to reach infections within the cells, while we class this and other drugs as germicides, we do not yet know whether they act purely as such or whether they do not also stimulate the cells to defensive activity in the manner that we believe antigens do.

Paper: "Reflex Vasomotor Changes in the Nasal Mucous Membrane."

By A. L. Tatum, M. D. (By Invitation).

ABSTRACT.

In recording vasomotor changes in the nasal mucous membranes of dogs and rabbits by means of the plethysmographic method described by Tschalussow in 1913, the speaker observed a vasoconstriction during or following partial asphyxia either by tracheal obstruction or by rebreathing of air containing CO_2. A vasodilation was observed on artificial respiration sufficient to produce a slight deficiency of CO_2 in the

blood. The vasoconstriction from partial asphyxia was prevented by section of the cervical sympathetic nerves, while by contrast the dilation from acapnia seemed to be unchanged.

Subjective experiments by the speaker and others seemed to confirm the above mentioned findings of nasal vasoconstriction or a sensation of diminished nasal resistance to air passage after mild asphyxia produced by rebreathing from a rubber bag or by holding of the breath for a short time, and vasodilation or a sense of increased nasal resistance on voluntary overventilation.

Objective experiments were devised in such a manner as to avoid irritation of the nasal passages. By leading a constant stream of air under low positive pressure through a rubber tube to one nostril air will be forced into the corresponding nasal chamber and find its way out through the mouth, or, if the mouth and glottis be closed, out through the other nasal passage. A side tube is connected to a tambour or other recording device so as to record the lateral pressure within the rubber tube carrying the air to the nasal chamber. Obviously the tube inserted into the nostril must fit closely so as to avoid variable air leaks and also changes in position of the ala. By such a device the speaker's interpretation of the subjective sensations of resistance to air passage were confirmed by objective measurements.

<div align="center">DISCUSSION.</div>

DR. A. J. CARLSON thought it was really no use for him to discuss this paper, because Dr. Tatum knew as much about the subject of vasomotor reflexes of the nose as he did, or more, as he had been working in this field for some time while Dr. Carlson had not. He was proud of Dr. Tatum's work. The results may not have at present any important clinical bearing but they extend our knowledge. Dr. Carlson thought the first result (vasoconstriction or slight asphyxia) was not difficult to interpret. We know that partial asphyxia produces vasoconstriction in general. This, according to Dr. Tatum's work, includes the nasal mucous membrane. The opposite results (vasodilation by low CO_2 in the blood) were not so readily explained, and that meant that Dr. Tatum or someone else would have to go ahead and see what happens in other organs

under this condition. They will have to put the arm, the leg and other organs in the platysmograph and see if, when the CO_2 content of the blood is greatly reduced, general vaso-dilatation follows. This is probably the case.

Of course, it is perfectly obvious that vasoconstriction in the membrane will allow more air to go in if the other passages are open, and it is obviously advantageous to secure more air in partial asphyxia, but Dr. Carlson could not see the advantage on the other side. The partial blocking of the nares by turgescence would be of no great value in preventing further ventilation. Dr. Tatum's work was a significant contribution in a field of growing importance. Everyone knew the recent developments in the field of vasomotor control, particularly the work of Dr. Krogh on the capillaries, with the possibility that the capillaries themselves are under control of vasomotor nerves and that in addition there is locally or peripherally an axone reflex. The vasomotor reflexes that interested this audience more particularly in connection with the disturbances in the nose and middle ear were the local ones.

DR. NORVAL H. PIERCE said he thought he was asked to be present to listen and see if the facts deduced by Dr. Tatum could have practical clinical application, but he could see but a very limited application to the pathologic conditions encountered in the everyday work of the laryngologist and rhinologist. He believed it would be somewhat difficult to induce a patient to wear plugs of cotton in the nose that was already stopped up by the turgescence of hay fever. He was inclined to believe that the hay fever and socalled angioneurotic rhinitis cases, which, he might add parenthetically, are undoubtedly rapidly increasing in numbers, have to do largely with the peripheral reflex mechanism. His reason for this was that in these cases with turgescence of the turbinates the excretion of large amounts of colorless mucus and attacks of paroxysmal sneezing, amelioration of the symptoms may be obtained by cauterizing the sensitive areas of the nose—**the anterior head** of the middle turbinate body, the anterior head of the inferior turbinate body, and the tuberosity of the septum. Especially are the fits of sneezing reduced in frequency and violence, which indicates that we influence a smaller reflex arc than the

one with which Dr. Tatum has dealt. Unfortunately, in almost all other pathologic conditions in the nose there are constant plugs in the nose, unchanging plugs in the nose; for example, the socalled mulberry hypertrophies of the posterior ends of the turbinates. They do not contract very much, especially when connective tissue is present and when they assume the mulberry stage that has already occurred. Where there is the true hyperplasia of the turbinate bodies, or when the nose is plugged up with polypi there is nasal asphyxia—stoppage of the nose—but these tissues cannot contract. They remain permanently as plugs in the nose. It has been said that hay fever patients when they go into an icebox and remain there for some time have contraction of the nasal mucosa and breathe very much better for some time after they come out. That may be explained by assuming that the stimulation of the cold air causes constriction of the nasal blood vessels.

Dr. Pierce expressed his profound appreciation of Dr. Tatum's very beautiful piece of work and said it represented the kind of work that permanently advances medicine.

Dr. J. Holinger asked whether Dr. Tatum made the opposite experiment with negative pressure. This experiment is being made nearly every day in his office, Dr. Holinger said. It is the suction treatment of diseases of the nose. The invariable result is a great relief of the stuffiness and congestion, and the nose is much freer and clearer for many hours afterward, although it sounds very paradoxical that suction, although it is done to produce a hyperemia, relieves the hyperemia.

Dr. Edwin McGinnis thought that a practical point to be gained from this experiment was that it is well not to do anything to the nasal mucosa that will inhibit the action. The old method of cauterization, cutting off the lower border of the inferior turbinate, handicapped the physiologic action of the nose, and this experiment of Dr. Tatum's showed that that type of work is not very good from the physiologic standpoint.

Regarding hay fever and the fact brought out by Dr. Pierce, that the hay fever patient is able to breathe better after being in an icebox for some time, Dr. McGinnis thought this had been noted by everyone. His explanation was that in the icebox there was no pollen, and if the patient remained in there

long enough the reaction subsides and the mucosa again takes
up the normal function, with the result that the patient has
good breathing until they get more pollen in the nose, when
they have another upset. It has been noted in these patients
that after a good rain that washes away the pollen and dust,
the patients are much better until we get a south wind that
fills the air again. It is also known that in this locality, when
there is an east wind blowing off the lake, these patients are
much more comfortable, which seemed to Dr. McGinnis to
prove that this is more a question of infection and not vaso-
motor reaction.

Dr. McGinnis thanked Dr. Tatum for bringing this subject
before the society, for it emphasized the idea that it is not
well to do much destructive surgery to the nasal mucosa.

Dr. Alfred Lewy asked if anything in Dr. Tatum's experi-
ments threw any light on the commonly observed phenomenon
of nasal obstruction alternating sides.

Dr. J. Gordon Wilson expressed his pleasure at listening
to this very interesting paper. The understanding of the
influence of the nervous system on the mucous membrane of
the nose and on the nasal capacity is of fundamental impor-
tance to rhinologists. Hence, they welcome any physiologic
light that can illumine their ignorance. When it is recalled
that the nasal capacity and respiration can be modified by a
nervous system extending from the fifth cranial nerve to the
lumbar plexus, when one adds to this cerebral stimuli—emo-
tions (fear)—which apparently modify nasal capacity as well
as respiratory rate and depth, one is impressed with the wide
source of respiratory influences. Luckily this wide range is
not constantly in activity. The vagus and sympathetic and
dorsal nerves apparently can do the greater part of what is
required. Yet the adjunct system can operate and may oper-
ate to our disadvantage. It may destroy the coordination
through which the normal pathways act.

The technic on which the results presented were based has
advantages over those of the Russian observer. The addition
of a tambour will give readings more easily read. The con-
stant stream of air, if it can be made effective, is a decided
step forward. It has always seemed to him that to base de-
ductions in regard to variations in nasal capacity from the

sensation of diminution of nasal resistance, following voluntary apnea, is open to the following objections:

1. The inspirations following the voluntary cessation of breathing is a compensatory dyspnea, and the air is taken in with greater negative pressure.

2. On account of the increased negative pressure the air pathway through the nose is altered. Therefore one has increased suction plus altered nasal relations.

3. Reactions arising from the glottis give sensations difficult to eliminate.

There objections would appear to be obviated by a constant stream of air at a fixed pressure.

The anatomy of the turbinates shows a structure designed so as to respond rapidly and with great mass variations to respiratory needs and therefore is a mechanism adaptable for nerve reflex stimuli. The venous spaces with their thick walls of muscular and elastic tissue readily and quickly react to stimulation. But is the stimulation always reflex from the periphery? What about the stimuli which arise directly in the respiratory and the vasomotor centers from changes in the circulating blood? The activity from the cerebrum he had already referred to. It appeared to him that one has also to consider local changes in the nasal vessel walls in response to the circulating blood in the venous sinus, and it may be influenced by absorption from surface epithelial cells. Can such a peripheral factor be disregarded?

Clinically it is frequently desired to secure permanent volume changes in nasal capacity. One of the persistent symptoms of a continued rhinitis is a weakening in the tonicity of the vessels of the turbinates, resulting in a fluctuating or more or less persistent mass obstructing breathing. The discussion pointed out how this hypotonicity may be eliminated by copying physiologic means of constriction.

The exposure of the respiratory center to widespread afferent impulses, to cerebral influences, as well as to conditions in the circulating blood, awakened one's admiration of the mechanism which blends the incoming impulses so harmoniously into the two factors of respiration. Should the blending fail, the rhinologist may be asked to find the defective factor. Dr. Tatum's paper pointed out some of these factors and

suggested others—and for this reason, as well as others, it was a pleasure to have it read.

Dr. A. J. Carlson said that probably the remarks he was going to make should be left to Dr. Tatum, but he was older and more cruel than Dr. Tatum, who would probably not make them.

In the discussion of this physiologic paper, where every step had been proved, Dr. Carlson was grieved that some of the gentlemen had talked too lightly of matters of theory as if they were demonstrated facts. He wished to know what the speakers or anyone else had done to show that the pathologic conditions described were due to focal reflexes, and therefore he thought everyone should talk with more hesitation until this had been done. If we keep repeating an hypothsis often enough some of us think the hypothesis is a demonstrated fact, and the "urge" to replace theory by demonstrated fact is lost.

Dr. J. Gordon Wilson asked Dr. Carlson if it were not the case that we could have in the venous channel of the nose a local reaction of dilatation or constriction brought about through changes in the blood circulating there.

Dr. Carlson replied that this was true under certain conditions. That fact and the proof that that kind of reaction is involved in any one of the phenomena described by Dr. Tatum was certainly taking for granted many things that have not been proven. He believed that the gentlemen had assumed too much, that they could hypnotize themselves and that if they repeated this long enough they would think it had been proven.

Dr. Norval H. Pierce said that Professor Carlson was not brutal but platonic. Plato said that "the just retribution for him who errs is that he be set right." That is not brutal. There was no doubt that those who had discussed the paper were not physiologists. There is a good deal of loose thinking even among physiologists, but medical men had to think, they had to attempt to reason in their work, especially if the physiologists had been so dilatory in their work as not to have made all things clear.

Dr. Tatum (in closing) said in regard to the pathology of the nose that he would make a parallel to inflammation elsewhere. It is well established, he thought, that inflamed tissue

involves blood vessels which are relatively irresponsive to vasoconstrictor agencies. He was not sure that these reflexes he had described could be elicited equally well in pathologic conditions and admitted that he did not know anything about it. They were not clinicians and had not the opportunity to study such conditions, so he would not say that the pathology might not alter the responsivity.

In regard to the negative pressure, that seemed logical, and he would be glad to try it out. He thought he might have misled the gentlemen in his remarks, for he had not used positive pressure to any extent, using very slight positive pressure only.

Regarding the reflex action, all Dr. Tatum wished to say was that this method of plethysmographing a viscus is the most sensitive of any he had had the good fortune to try. It is easily established and exceedingly sensitive. In his hands, in animals that were anesthetized, he obtained no response after section of the cervical sympathetics, indicating a necessary nervous reflex. Of course, these animals were abnormal when they were anesthetized, and what they would have done had they been conscious and normal he did not know.

Dr. Tatum said he had thought about the alternate changes a good deal. One side of the nose will be blocked up and the other side free, and then a reversal. That might be a nervous affair. One individual had reported to him after he had heard of this work that he had tried partial asphyxia because he was subject to intumescent rhinitis. He tried it at night and obtained relief for a considerable length of time.

In regard to the question pertaining to the effects of want of air after one had held his breath for a time, Dr. Tatum said this was not immediately evident. The increased patency was not evident until after the lungs were fairly well ventilated again. It was his habit to breathe normally for several breaths until he did not feel air hunger and was no longer asphyxiated. Three or four breaths would usually suffice, after which objective or subjective measurements are safely made. If he breathed harder and respired a greater volume of air, it was difficult to compare the resistances, for obviously resistance is a function of both rate-volume of air flow and of diameter of passage.